TO STUDY THE RELATIONSHIP OF AMNIOTIC FLUID VOLUME WITH NEONATAL RESPIRATORY MORBIDITY

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I hereby declare that the thesis entitled "**TO STUDY THE RELATIONSHIP OF AMNIOTIC FLUID VOLUME WITH NEONATAL RESPIRATORY MORBIDITY**" submitted towards partial fulfillment of the requirements for the award of **MD in OBSTETRICS & GYNAECOLOGY** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Pooja Tandon**, Associate Professor, Department of Obstetrics & Gynaecology, **Dr. Ashima Taneja**, Professor & Head, Department of Obstetrics & Gynaecology and **Dr. Kamaldeep Arora**, Associate Professor, Department of Pediatrics. No part of this thesis has formed the basis for the award of any degree previously.

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Dedicated

to

my Teachers

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Family

LIST OF ABBREVIATIONS

ACOG	AMERICAN COLLEGE OF OBSTETRICS AND GYNAECOLOGY
AFI	AMNIOTIC FLUID INDEX
AOR	ADJUSTED ODDS RATIO
BMI	BODY MASS INDEX
CI	CONFIDENCE INTERVAL
CPAP	CONTINUOUS POSITIVE AIRWAY PRESSURE
CRP	C REACTIVE PROTEIN
CS	CESAREAN SECTION
ENaC	EPITHELIAL SODIUM CHANNELS
fFn	FETAL FIBRONECTIN
FIRS	FETAL INFLAMMATORY RESPONSE SYNDROME
HVS	HIGH VAGINAL SWAB
IL	INTERLEUKIN
LBW	LOW BIRTH WEIGHT
MMP	MATRIX METALLOPROTEINASE
MSAF	MECONIUM STAINED AMNIOTIC FLUID
Na+K+ATPase	SODIUM-POTASSIUM ADENOSINE TRIPHOSPHATASE
NEC	NECROTISING ENTEROCOLITIS
NICU	NEONATAL INTENSIVE CARE UNIT
P	PEARSON'S COEFFICIENT
PCR	POLYMERASE CHAIN REACTION
PGE2	PROSTAGLANDIN E2
PPROM	PRETERM PREMATURE RUPTURE OF MEMBRANES
RDS	RESPIRATORY DISTRESS SYNDROME
RR	RELATIVE RISK
sPL with IM	SPONTANEOUS PRETERM LABOR WITH INTACT MEMBRANES
TIMP	TISSUE INHIBITOR OF MATRIX METALLOPROTEINASE
TNF-alpha	TUMOUR NECROSIS FACTOR alpha
TTNB	TRANSIENT TACHYPNEA OF NEWBORN
UTI	URINARY TRACT INFECTION

ABSTRACT

Introduction: Preterm premature rupture of membranes (PPROM) is defined as spontaneous rupture of fetal membranes before 37 completed weeks. The major pre disposing event is intra-uterine infections. PPROM complicates approximately 3 percent of pregnancies and leads to one third of preterm births.

Aims & Objectives: To determine the effect of preterm premature rupture of membranes on neonatal respiratory morbidity.

Material & Methods: A prospective clinical study was conducted in the department of Obstetrics and Gynaecology at Dayanand Medical College &Hospital, Ludhiana (Punjab) for a time period of 2 years, from September 2019 to September 2021. The study was performed on 105 patients admitted to the labour ward of DMCH with PPROM between 24 weeks – 36weeks 6 days of gestation. Out of 105 PPROM patients, 5 were lost to follow up, rest 100 women were followed up till delivery for the neonatal outcome.

Results: The mean age of the patients presenting with PPROM was 29 years and the mean gestational age at presentation was 32+/- 3weeks. The majority of patients were educated and booked cases. The most common risk factor in patients with PPROM was genito-urinary infection followed by a history of prior preterm birth. The mean duration of leaking per vaginum at the time of admission to the hospital was 22hours and half of the patients went into labour within 24 hours of membrane rupture. Induction of labour was required in 45% patients and 50% underwent caesarean section for obstetric indications. Majority of babies in our study were low birth weight (88%). Low Apgar scores and more NICU admissions were seen in extremely preterm neonates. Respiratory distress syndrome, congenital pneumonia and transient tachypnea of newborn were seen mostly in extremely preterm, very preterm and late preterm neonates respectively.

Conclusion: It was observed that the risk of neonatal respiratory complications changes with gestational age of PPROM and delivery. Respiratory distress was the most common complication. Risk scoring strategies involving the demographic variables along with previous history of preterm deliveries should be developed to identify the high risk cases and treat them prior to rupture of membranes.

Keywords: PPROM-preterm premature rupture of membranes, RDS-respiratory distress syndrome, TTNB-transient tachypnea of newborn, Congenital pneumonia

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INTRODUCTION

Preterm premature rupture of membranes (PPROM) is defined as spontaneous rupture of fetal membranes before 37 completed weeks of gestation, the major predisposing event being intra-uterine infections. PPROM complicates approximately 3 percent of pregnancies and leads to one-third of preterm births.[1]

The risk factors associated with PPROM are low socioeconomic status, body mass index (BMI) <19.8, nutritional deficiency and cigarette smoking. Women with preterm premature rupture of membranes in the previous pregnancy are at increased risks for recurrence during a subsequent pregnancy. In most cases of preterm rupture, however, no known risk factor is identified.[2]

Contributing factors:

Various genetic and environmental factors affect the frequency of preterm labor. Vaginal bleeding in early pregnancy i.e. threatened abortion can be associated with preterm labor, placental abruption and subsequent pregnancy loss before 24wks.[3] Young or advanced maternal age, poverty, short stature, vitamin C deficiency and obesity in mothers are implicated in causing preterm birth. Working long hours, hard physical labour and psychological factors like depression, anxiety and chronic stress have been reported in association with preterm birth and PPROM.

Genetics may also play a causal role as suggested by the recurrent familial and racial nature of preterm birth and PPROM. Intervals between pregnancies less than <18 months and >59 months can be associated with preterm birth.

A major risk factor for preterm labor and PPROM is prior preterm delivery. The risk of recurrence is around three times compared with that of women whose first neonate was born at term. It has been seen that the causes of prior preterm delivery can also recur in the subsequent pregnancy.

Infections cause a considerable proportion of PPROM cases. It is estimated that 25-40% of preterm births result from intrauterine infections. They activate the innate immune system. Microorganisms bring about the release of inflammatory cytokines such as interleukins and TNF-alpha. These cytokines stimulate the production of prostaglandins like prostaglandin E and matrix degrading enzymes. Prostaglandins stimulate uterine contractions. Degradation of the extracellular matrix in the fetal membranes, brought about by matrix degrading enzymes, leads to preterm rupture of membranes.[4]

Complications:

Among women with preterm PROM, clinically evident amnionitis occurs in 15–35% of cases and postpartum infection occurs in approximately15–25% of cases. Abruptio placentae complicate 2–5% of pregnancies with preterm PROM.[1] Generally, amnionitis and endometritis respond to parenteral antibiotic therapy , rare but serious complications such as maternal sepsis and death may also occur.

The neonatal outcome following prolonged preterm rupture of membranes is compromised. It increases the risk of prematurity . The preterm infant is susceptible to a range of medical complications during the newborn period as well as morbidities extending later into life including a 1 to 2 % risk of fetal death. These complications can be respiratory distress syndrome, pneumothorax, bronchopulmonary dysplasia, pneumonia, necrotizing enterocolitis, intraventricular hemorrhage and retinopathy of prematurity. The most common complication among these is respiratory distress syndrome (RDS).

Respiratory distress syndrome is caused by a deficiency of surfactant. Surfactant is synthesized by type II pneumocytes. It is essential to stabilize the air expanded alveoli. Inadequate surfactant, as seen in cases of RDS, leads to the formation of hyaline membranes in the distal bronchioles and alveoli. These membranes are composed of fibrin-rich protein and cellular debris. The result is necrosis of epithelium underlying the membranes because of which, fetal lungs are unable to sustain necessary oxygenation. The resulting hypoxia compromises the development of both airways and

pulmonary vasculature in the fetus. Adaptation to extrauterine life is also impaired.[5]

Management:

Gestational age is a primary factor when considering management in patients of PPROM.Patients with preterm PROM before 34 weeks of gestation are managed expectantly if no maternal or fetal contraindications exist.[6,7] The clear indications for delivery are abnormal results from fetal heart rate testing, clinical intra-amniotic infection, and significant abruptio placentae. Expectant management of preterm PROM generally consists of hospital admission with periodic assessment for infection, abruption, umbilical cord compression, fetal well-being and onset or progress of labor. A temperature elevation may indicate intrauterine infection. Prompt diagnosis of intraamniotic infection in preterm pregnancy has to be made because early signs and symptoms may be subtle. For diagnosing infection in the absence of fever, other clinical criteria, such as abdominal or fundal tenderness and maternal or fetal tachycardia have to be looked for. If antenatal corticosteroids have been administered then serial monitoring of leukocyte counts and other markers of inflammation is not useful and is nonspecific when there is no clinical evidence of infection.[8]

Tocolytic agents are considered in preterm PROM for steroid benefit to the neonate, especially at earlier gestational ages, or for maternal transport to a healthcare center. They are used cautiously and avoided if there is evidence of infection or abruption. Tocolytic therapy is not recommended in the setting of preterm PROM between 34 weeks and 36 weeks 6 days period of gestation.[1]

A single course of corticosteroids is recommended for pregnant women between 24 weeks of gestation and 33 weeks 6 days of gestation. They are considered for pregnant women who are at risk of preterm birth within 7 days even at 23 weeks of gestation if they present with ruptured membranes.[9,10] A single repeat course of antenatal corticosteroids is considered in women with preterm PROM who are less than 34 weeks of gestation, are at risk of preterm

delivery within 7 days, and whose prior course of antenatal corticosteroids was administered more than 14 days back. However, delivery is not delayed to achieve a rescue course.[10]

Administration of broad-spectrum antibiotics prolongs pregnancy and reduces maternal and neonatal infections. It also decreases gestational age-dependent morbidity.A 7-day course of latency antibiotics with a combination of intravenous ampicillin and erythromycin followed by oral amoxicillin and erythromycin is recommended by ACOG during expectant management of women with preterm PROM who are at less than 34 weeks of gestation.[11]

Delivery has traditionally been recommended for all women with ruptured membranes after 34 weeks of gestation.The decision regarding expectant management or immediate delivery in patients with PROM between 34 weeks of gestation and 36weeks 6 days period of gestation is made after taking both maternal and neonatal perspectives into consideration. Care is individualized through shared decision-making, and expectant management is not extended beyond 37 weeks of gestation in cases of PPROM.[1]

Preterm birth and PPROM remain an important public health priority worldwide. Evidence-based strategies to prevent prematurity from occurring as well as mitigating its effects on preterm neonates are needed, particularly in low-resource settings.

AIMS & OBJECTIVES

The aim of the study is to determine the effect of preterm premature rupture of membranes on neonatal respiratory morbidity.

REVIEW OF LITERATURE

Amniotic fluid provides a protective environment for the fetus during pregnancy and labour. Preterm premature rupture of membranes is defined as spontaneous rupture of fetal membranes before 37 completed weeks and before labor onset. At term, prelabour rupture of membranes complicates 8% of pregnancies. It is mostly followed by the onset of spontaneous labour and delivery.[12] While with preterm premature rupture of membranes, birth within 1 week of rupture of membranes occurs in at least one-half of patients regardless of obstetric management or clinical presentation.[13] Preterm premature rupture of membranes complicates approximately 3 percent of pregnancies and leads to one-third of preterm births.[1]

AMNIOTIC FLUID- COMPOSITION AND REGULATION

Early in pregnancy, the amniotic cavity is filled with a fluid that is similar in composition to the extracellular fluid. During the first half of pregnancy, transmembranous flow occurs across the amnion, intra-membranous flow occurs across the fetal vessels on the placental surface and transfer occurs across the fetal skin. Fetal urine production begins between 8-11 weeks. It becomes a major component of amniotic fluid in the second trimester. Until keratinization occurs at 22 - 25 weeks, water transport across the fetal skin continues.[14]

With advancing gestation four pathways play a key role in amniotic fluid volume regulation. 1) **Fetal urination** is the primary source of amniotic fluid . By term fetal urine production may exceed upto 1 liter per day and the entire amniotic fluid volume is recirculated on a daily basis.2) the osmolality of maternal and fetal plasma is approximately 280 mOsm/ml and that of Amniotic fluid is 260 mOsm/ml. This makes the amniotic fluid hypotonic. This accounts for significant **intra-membranous fluid transfer** (400ml/day) across and into the fetal vessels present on the placental surface.[15] 3) the Third source of regulation of amniotic fluid is the **respiratory tract**. Approximately 350ml of lung fluid is produced daily in the later part of gestation and half of this is immediately swallowed. 4) **Fetal swallowing** is the

primary mechanism for amniotic fluid resorption . It averages about 500-1000ml per day.[15]

PATHOGENESIS OF PRETERM PREMATURE RUPTURE OF MEMBRANES

Membrane rupture at term is a normal part of parturition and can occur before or after the onset of contractions. This results from a combination of cellular apoptosis (programmed cell death), increased collagenase activity and dissolution of the amniochorionic extracellular matrix. All these can be exacerbated by contraction-induced shearing forces.

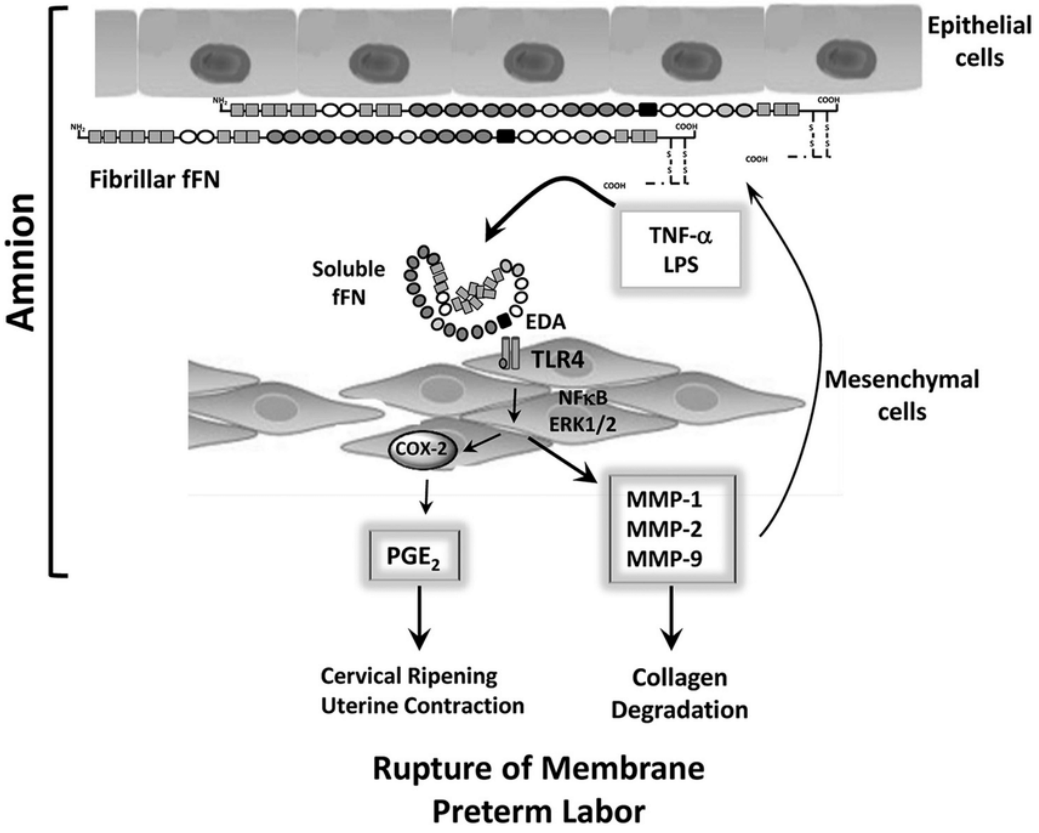
The amnionic extracellular matrix and interstitial amnionic collagens-primarily type I and III provide the tensile strength of the membranes. These are produced in mesenchymal cells.[16] The matrix metalloproteinase (MMP) family is involved with normal tissue remodeling .They are particularly involved with collagen degradation. The regulation of MMP activity is done by tissue inhibitors of matrix metalloproteinases or TIMPs. The MMP-1, MMP-2 , MMP-3 And MMP-9 members of this family are found in elevated concentrations in the amniotic fluid from pregnancies with PPROM. TIMPs are found in lower concentrations in these pregnancies. The expression of MMPs can be increased by treatment with IL-1, TNF-alpha and IL-6.[17]

Another mechanism has been studied for the occurrence of PPROM. According to this, bacterial endotoxin or TNF-alpha elicits the release of fetal fibronectin (fFn) by amnion epithelial cells. The fFn then binds to toll-like receptor 4 in the amnion mesenchymal cells and activates signaling cascades. These result in increased prostaglandin E synthesis (PGE2). Increased prostaglandin levels promote cervical ripening and uterine contractions. This also elevates the activity of MMP-1, MMP-2 and MMP-9. Increased MMPs allow collagen breakdown in the fetal membranes resulting in premature rupture.[18]

The amnion exhibits a higher degree of cell death and more apoptosis markers than that in term amnion in pregnancies with PPROM. Apoptosis is possibly regulated by bacterial endotoxin, IL-1 and TNF-alpha. Lastly, there are proteins involved in the synthesis of mature

cross-linked collagen or matrix proteins that bind collagen and thereby help in promoting tensile strength. These proteins are altered in membranes with premature rupture.[19]

Thus, PPROM is a pathological condition with numerous etiologies and causes resulting from collagen degradation, altered collagen assembly as well as cell death. All these lead to a weakened amnion. Bacterial cultures of amniotic fluid support the role of infection in a significant proportion of cases.



PICTURE 1: Model of fFN signaling in the pathogenesis of PPROM and preterm labor.[20]

MATERNAL AND FETAL COMPLICATIONS ASSOCIATED WITH PPROM.

MATERNAL COMPLICATIONS:

1. **Chorioamnionitis**

25% of all cases of PPROM develop chorioamnionitis. The clinical diagnosis of chorioamnionitis requires the presence of fever (>100º F or 37.80º C)

and two or more of:

- Maternal pulse > 100 bpm
- Fetal heart rate > 160 bpm
- Abdominal tenderness
- Foul-smelling vaginal discharge
- Leukocytosis > 15000
- C-reactive protein >2.7 mg/dl
- with no other infectious site involved.

The risk of intrauterine infection increases with the duration of membrane rupture and with declining gestational age , due to decreased antibacterial activity of amniotic fluid remote from term.[21] Burchell et al[22] found that 1.7% of his patients developed fever within 2 hours, 7.5% between 24 and 48 hours and 8.6% beyond 48 hours. In another study, the prevalence of chorioamnionitis was 2.7% before 12 hours, 6.3% between 12 and 24 hours and 26.4% after 24 hours of latency.[23]

2.Placental separation and abruption

Placental abruption affects 4-12% of pregnancies with PPROM.[24]

Placental abruption and hemorrhage occur in 10% of the cases with PPROM secondary to uterine decompression and inflammation, as opposed to 1% of the general obstetric population. According to Rotschild et al,[25] placental abruption occurs in up to 50% cases of PPROM before 20 weeks of gestation.

3. Postpartum endometritis:

Postpartum endometritis is a frequent maternal complication in women with PPROM, particularly if they develop chorioamnionitis (40%) and are delivered by cesarean section. Shumway et al,[26] reported that the incidence of postpartum maternal sepsis was between 0% and 3%, but sepsis leading to death had an incidence of 0.14%.

4. Risk of operative delivery: Caesarean delivery rates significantly increase in PPROM, secondary to obstetric complications such as fetal malpresentations, non-reassuring fetal heart patterns, cord compression and placental abruption.

NEONATAL COMPLICATIONS

1. RESPIRATORY DISTRESS SYNDROME

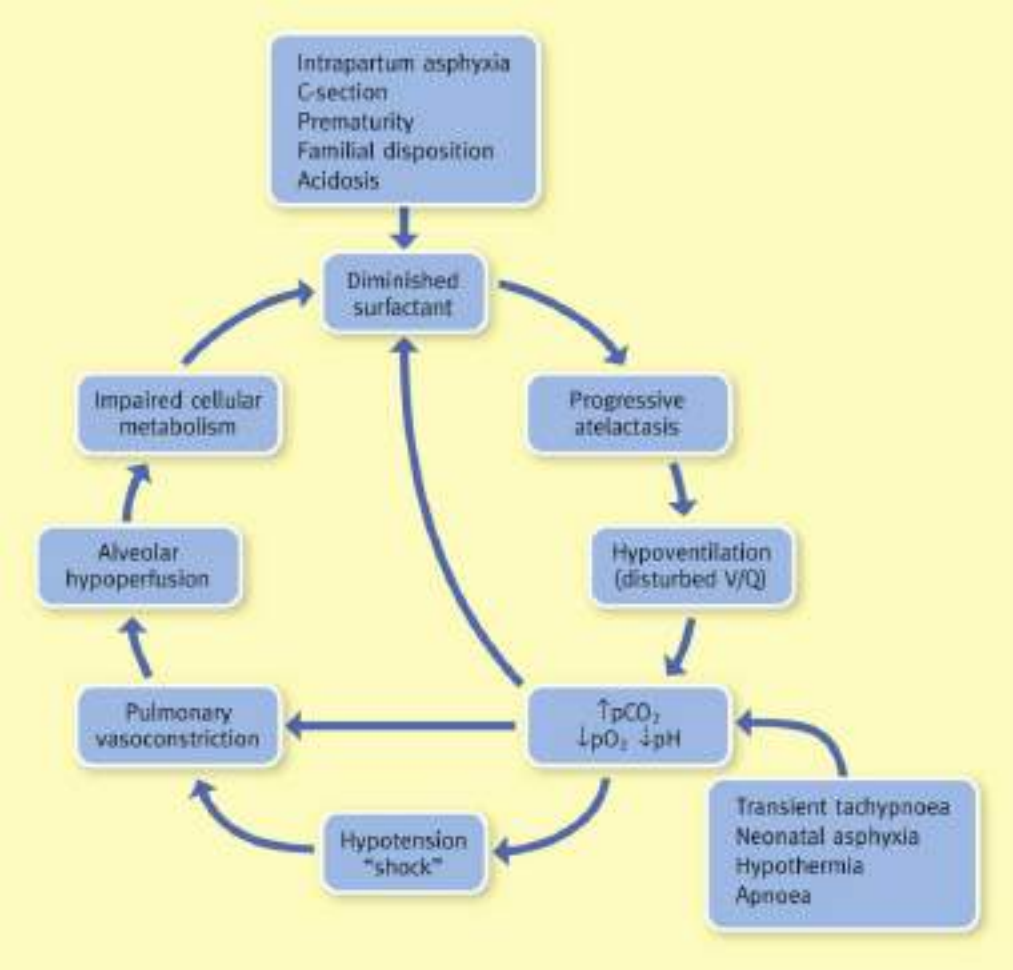
Respiratory distress syndrome (RDS) occurs primarily in premature infants. Its incidence is inversely related to gestational age and birth weight. It occurs in 60–80% of infants at < 28weeks gestational age and reduces to 15-30% at 32 – 36 weeks of gestation. Maternal diabetes, multiple births, cesarean delivery, asphyxia, cold stress, and a maternal history of previously affected infants increase the risk for the development of RDS. The risk of RDS is reduced in pregnancies with chronic or pregnancy-induced hypertension, maternal heroin use, prolonged rupture of membranes, and antenatal corticosteroid prophylaxis.[27]

Respiratory distress in a newborn is identified by tachypnoea i.e. respiratory rate of >60/min, lower chest retraction and grunting. It is due to breathing out through a partially closed glottis which generates enough pressure inside the lungs so as to prevent alveolar collapse at the end of expiration.

Pathophysiology:- Surfactant is synthesized by type II pneumocytes in the lungs. Surfactant is essential to stabilize the expanded alveoli. It lowers the surface tension and prevents lung collapse during expiration. When the surfactant is inadequate, alveoli are unstable and low-pressure causes collapse at end-expiration. The nutrition of pneumocytes is compromised by hypoxia and systemic hypotension. Partial persistence of fetal circulation may lead to pulmonary hypotension and a relative right to left shunt. Eventually, alveolar cells undergo ischemic necrosis. When oxygen therapy is initiated, the pulmonary vascular bed dilates and the shunt reverses. Protein-filled fluid leaks into the alveolar ducts. This leads to sloughing of the cells lining the ducts. Hyaline membranes composed of fibrin-rich protein and cellular debris line the dilated alveoli and terminal bronchioles. The epithelium underlying the membrane becomes necrotic.

Shunting of blood through non-ventilated lung leads to hypoxemia and metabolic and respiratory acidosis. The chest radiograph shows diffuse reticulogranular infiltrates and an air-filled trache-bronchial tree known

as air bronchogram.[5] If surfactant is given and / or early continuous positive airway pressure (CPAP) is applied, the clinical course is altered and recovery is faster.



PICTURE 2: Pathophysiology of respiratory distress syndrome[27]

2. TRANSIENT TACHYPNEA OF THE NEWBORN(TTNB)

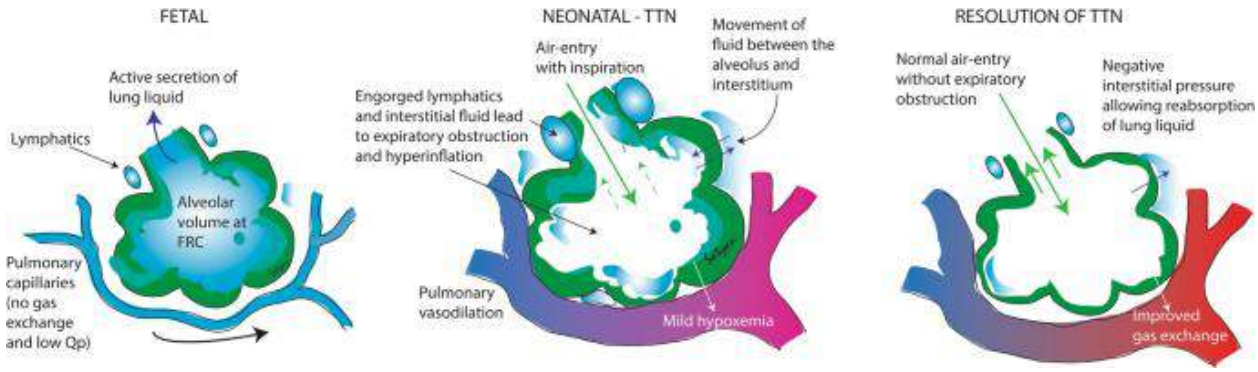
Transient tachypnea of the newborn (TTNB) is associated with delayed clearance of fetal lung fluid. It is a clinical syndrome of self-limited tachypnea. Although the actual incidence is likely underreported, it is estimated at 3-6 per 1,000 term infant births. This makes TTNB the most common etiology of tachypnea in the newborn. Common associated risk factors are twin gestation, maternal asthma, late prematurity, precipitous delivery, gestational diabetes, and cesarean delivery without labor.[28]

Pathophysiology: Clearance of fetal lung fluid occurs through increased expression of epithelial sodium channels (ENaC) and sodium-

potassium adenosine triphosphatase (Na+K+ATPase). These channels drive active sodium (as well as fluid) reabsorption. TTNB is believed to result from ineffective expression or activity of ENaC and Na+ K+ ATPase. It slows the absorption of the fetal lung fluid and results in decreased pulmonary compliance and impeded gas exchange.[27]

TTNB is characterized by the early onset of tachypnea (>60 breaths/ min), sometimes with retractions or expiratory grunting and occasionally with cyanosis that is relieved by minimal oxygen supplementation (<40%).

The chest generally sounds clear without crackles or wheeze. The chest radiograph shows prominent perihilar pulmonary vascular markings and fluid in the intralobar fissures. Rarely small pleural effusions may be seen. Hypercapnia and acidosis are uncommon. Most infants recover with supportive care alone. The tachypnea and oxygen requirements slowly resolve over the first 24-72 hours.[27]


FETAL
Active secretion of lung liquid
Lymphatics
Alveolar volume at FRC
Pulmonary capillaries (no gas exchange and low Qp)
NEONATAL - TTN
Air-entry with inspiration
Movement of fluid between the alveolus and interstitium
Engorged lymphatics and interstitial fluid lead to expiratory obstruction and hyperinflation
Pulmonary vasodilation
Mild hypoxemia
RESOLUTION OF TTN
Normal air-entry without expiratory obstruction
Negative interstitial pressure allowing reabsorption of lung liquid
Improved gas exchange

PICTURE 3: Airway liquid retention and role of respiration. The fetal fluid-filled lungs do not participate in gas exchange but the lung volume approximates the functional residual capacity (FRC) of the air filled lung after birth. Following the delivery of the head and air breathing, the pressure from inspiration creates a pressure differential that promotes airway liquid to move into the lung tissue, which can raise interstitial pressure. High interstitial pressure at end-expiration may shift fluid back into the alveoli.[29]

3. CONGENITAL PNEUMONIA

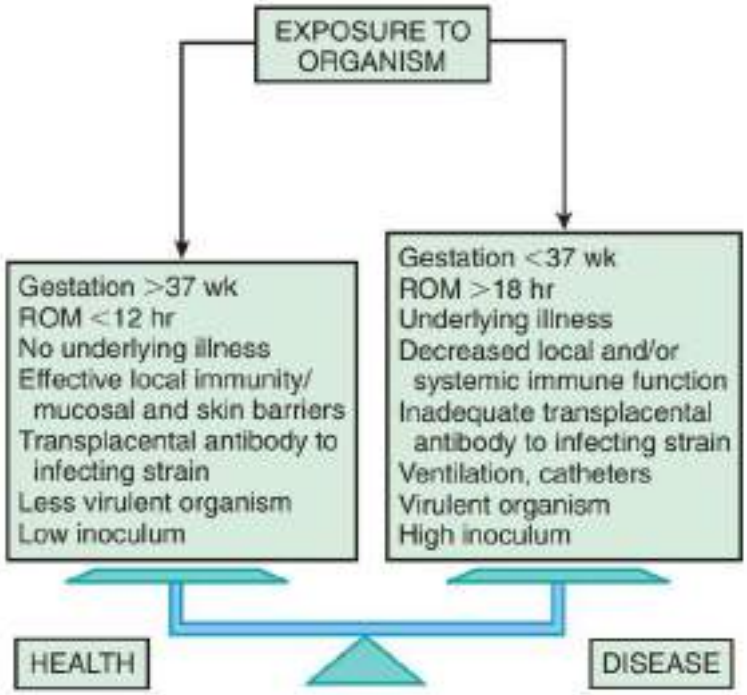
Congenital pneumonia is pneumonia with clinical and radiological evidence present at birth. Barton *et al.* found that congenital infection

which presented at ≤48 hours of age, was the most common (50.5%) cause of death in 111 extremely low birth weight infants (<1000g). They carried out an autopsy of these infants and also an examination of their placental and laboratory findings. They observed that congenital infection was significantly under diagnosed clinically. Most deaths were attributed to immaturity or respiratory distress syndrome. Moreover, they found that the predominant type of congenital infection affecting 53.6% of this series of extremely low birth weight infants was congenital pneumonia. They also found that the major cause of death was infection of the amniotic fluid leading to pneumonia.[28]

Risk factors and Causative organisms: Several risk factors have been identified which are associated with congenital pneumonia. These include maternal systemic infection, maternal chorioamnionitis , prolonged rupture of the chorioamniotic membranes and premature rupture of membranes. Clinical manifestations of congenital pneumonia may become apparent before delivery in the form of fetal distress or tachycardia. They may also become evident at delivery as a low Apgar score or severe respiratory distress. It is seen that in some infants, there may be a latent period of a few hours or an interval of 1-2 days before respiratory distress or shock develops.[28] In preterm infants, these signs of progressive respiratory distress may superimpose on respiratory distress syndrome. In some infants, the early signs and symptoms may be nonspecific. They may present with poor feeding, lethargy, irritability, cyanosis, temperature instability or with an overall impression that the infant is just not well.[28]

The clinical course of congenital pneumonia is very variable, depending on the underlying causative organisms and the presence of co-infection. Fulminant infection is most commonly associated with pyogenic organisms such as Group B *Streptococcus* and *L.monocytogenes*. The affected infants present with rapidly progressive circulatory collapse, respiratory failure and death within the first 24 hours of life.[30,31] A more indolent but persistent congenital pneumonia is caused by chlamydia and mycoplasma. Depending on the underlying

causative organisms, infants with mild-to-moderate pneumonia recover within 1 to 3 weeks with appropriate treatment.



PICTURE 4: Factors influencing the balance between health and disease in neonates exposed to a potential pathogen in PPROM[27]

4. NEONATAL INFECTION.

Neonatal sepsis is a clinical syndrome characterized by signs and symptoms of infection with or without accompanying bacteremia in the first month of life.33% of neonatal deaths are caused by sepsis globally. In majority of cases, the causative organisms are bacterial in origin. The three most common bacteria responsible for sepsis in India and other developing countries are Escherichia coli, Klebsiella pneumonia and Staphylococcus aureus.[5]

5. NECROTISING ENTEROCOLITIS

This newborn bowel disorder has clinical findings of ileus and blood in the stool. There is usually radiological evidence of bowel wall gas derived from invading bacteria also known as pneumatosis intestinalis. The pathological process is the uncontrolled inflammatory response to

bacterial colonization of the preterm intestine . Resection is done in cases with bowel perforation.

6.NEUROLOGICAL DISORDERS

Preterm PROM is associated with early delivery and perinatal infection and it is a potential risk factor for long-term neurological morbidity. Cerebral palsy and cystic periventricular leukomalacia have been linked to the presence of amnionitis , which is commonly seen after preterm PROM.[32] Similarly, brain lesions such as periventricular leukomalacia and/or the subsequent development of cerebral palsy have been linked to the elevated amniotic fluid cytokines and fetal systemic inflammation, which may accompany or reflect maternal or fetal infection in cases of preterm PROM . All are strongly associated with adverse neurodevelopmental outcomes.[32]

PREDICTION OF PPROM – NEWER APPROACHES

PPROM is the largest identifiable cause of preterm birth. No optimal screening test is present currently for PPROM in low-risk asymptomatic patients. Accurate PPROM diagnosis is essential for proper patient management. Traditional approaches to the diagnosis of rupture of fetal membranes are now powered by scientific progress. Biomarkers have a major role to play in this area. The search for an accurate test is still going on. New and interesting parameters have been proposed in recent years to improve the prediction of these events so as to make rational and safe use of the available therapeutic strategies. This can improve the outcomes of newborns in terms of prevention of related morbidities in the short and long term. It will also help to optimize the use of resources.

Nunes et al. reviewed the literature on fetal membrane thickness and its potential use for the prediction of PPROM. The area of fetal membrane imaging has a potential to be used in the future to predict the risk of preterm birth. Emerging technologies such as shear wave elastography, optical coherence tomography and fusion MRI imaging can help in improved examination of the fetal membranes. Along with

advances in ultrasound technology, future studies may be able to identify characteristics of the fetal membranes that are predictive of PPROM so that adequate preventive strategies can be undertaken.[33]

Mehra et al. demonstrated that a shorter transvaginal cervical length and an amniotic fluid index of 5 cm or less predicted that delivery will occur within 7 days in women presenting with PPROM. The potential to remain undelivered at 7 days following cervical length assessment was greatly improved by the combination of an amniotic fluid index greater than 5 cm and transvaginal cervical length greater than 2 cm. These findings may be helpful for counseling and optimization of maternal and neonatal care in women with PPROM.[34]

A recent study by Ryu H.K. et al.[35] proposed that maternal C-reactive protein (CRP) and oxidative stress markers can be used as predictors of delivery latency in women experiencing PPROM. CRP, lipid peroxide, and oxygen radical absorbance capacity levels may be useful in predicting delivery within 3 days after PPROM. Gezer et al. identified urea and creatinine levels in cervicovaginal fluid as prospective predictors of PPROM as well as PPROM to preterm delivery interval. In multivariate logistic regression analysis, vaginal fluid urea and creatinine levels were found to be significant predictors of PPROM (both $P<0.001$) and delivery within 48 hours after PPROM ($P=0.012$ and $P=0.017$, respectively).[36]

Wang et al. recently examined the proteome profile of amniotic fluid and maternal plasma for their diagnostic and prognostic value in PPROM. Liquid chromatography coupled with tandem mass spectrometry-based proteomic techniques were used to examine the proteome profiles of amniotic fluid and maternal plasma. 12 of 540 unique proteins were chosen for further detection. Excellent diagnostic accuracy for PPROM was observed in Placental protein 14, with a respective sensitivity and specificity of 100% and 87.5% when the cut-off value was 0.008 μg/mL.[37]

Kemin et al. evaluated the diagnostic value of procalcitonin and CRP for the prediction of subclinical intrauterine infection in patients with PPROM. Both parameters have a good predictive value and can be

used as diagnostic indicators for PROM associated with chorioamnionitis. Procalcitonin is more suitable for pregnant women at 28weeks to 33+6 weeks. Furthermore, amniotic fluid interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α) seem to be good predictors for fetal inflammatory response syndrome . It may help in improving the clinical management of patients with PPROM. The non-invasive technique of sampling amniotic fluid from vaginal secretions facilitates daily measurements and bedside assessment of cytokines. In this respect , it is preferable to invasive amniocentesis.[38]

Various studies have been conducted to understand the pathophysiology of PPROM and its effects.Athayde et al. found that patients with PPROM had higher Matrix Metalloproteinase-9 (MMP-9) concentrations than those with preterm labor and intact membranes who were delivered at term. Women with microbial invasion of the amniotic cavity had higher median MMP-9 concentrations than did those without microbial invasion regardless of membrane status (preterm labor: 54.5 ng/mL, vs. <0.4 ng/mL and in PPROM patients 179. 8 ng/mL, vs. 7.6 ng/mL, P<0.001).[39]

Romero et al. found that fetuses with preterm PROM have increased concentrations of the enzyme MMP-9. It is implicated in the mechanism of membrane rupture . These fetuses were found to have lower concentrations of IL-1β, TNF - Receptor 1, and TNF - Receptor 2 than fetuses with preterm labor and intact membranes. The authors suggested that the role of the fetus in the genesis of preterm PROM also deserves consideration.[40]

DiGiulio et al. investigated the amniotic fluid from 204 subjects with PPROM with cultivation and polymerase chain reaction (PCR) methods. The prevalence of microbial invasion of the amniotic cavity was 34% by culture, 45% by PCR, and 50% by both methods combined. The number of bacterial species revealed by PCR (44 species-level phylotypes) was greater than that by culture where only 14 species could be identified. The relative risk for histologic chorioamnionitis was 2.1 for a positive PCR and 2.0 for a positive culture. A positive PCR was associated with a lower mean birth weight.

It was also associated with higher rates of respiratory distress syndrome and necrotizing enterocolitis (NEC).[41]

Lee T et al. conducted a study to ascertain the predictive value of antecedent preterm premature rupture of membranes for recurrent preterm premature rupture of membranes and preterm delivery rates in the next pregnancy. The rates of recurrent preterm premature rupture of membranes (16.7%) and preterm delivery (34.2%) in the 114 study group patients were substantially greater (odds ratio, 20.6; 95% CI, 4.7-90.2; and odds ratio, 3.6; 95% CI, 2.1-6.4) than noted background rates (0.96% and 12.5%) but considerably less than the recurrence rates of either preterm premature rupture of membranes or preterm delivery that were reported by others. The gestational age of preterm premature rupture of membranes in the index pregnancy affected neither the magnitude of risk nor the gestational age of recurrent preterm premature rupture of membranes or preterm delivery in the subsequent pregnancy. Stratification of outcome measures into three subgroups that were based on the gestational age of index preterm premature rupture of membranes demonstrated no significant differences in the incidence of preterm premature rupture of membranes or preterm delivery.[42]

Bouvier D et al. compared three groups which consisted of women without prematurity, women with spontaneous preterm labor with intact membranes (sPL with IM) and women with PPROM . 60 criteria were considered about characteristics, lifestyle, medical, gynecological, obstetrical history of mothers, medication during pregnancy, events at delivery, and complications in neonates. Of the 6968 women selected, (2.8%) presented with PPROM and (3.2%) presented with sPL with IM. The specific risk factors for PPROM were body mass index (BMI) <18.5 kg/m^2, history of PPROM, nulliparity, gestational diabetes, and low level of education. The complications associated with PPROM were abruption placentae, cesarean, APGAR at 5 minutes <4, birth weight <2500 g, stillbirth, neonatal jaundice, and prolonged hospitalization of mother and neonates. All these complications were also associated with sPL with IM. This study confirmed some of the risk factors of

PPROM and highlighted a new one which was gestational diabetes mellitus. Outcomes of PPROM were related to prematurity.[43]

In a study by Rana M. et al, a total of 150 pregnant women between 24 and 36 weeks gestation with premature rupture of membranes were subjected to detailed history and examination. Each patient was followed till her delivery. Feto-maternal outcome was recorded gestation-wise between 24 and 28 weeks (n = 15), 28 and 32 weeks (n = 30), 32 and 34 weeks (n = 90), and 34 and 36 weeks (n = 15). The mean latency period from membrane rupture to delivery decreased from 15 days at 24 to 28 weeks, 11 days at 28 to 32 weeks to 4.4 days at 32 to 34 weeks to 2.1 days at 34 to 36 weeks. The rate of spontaneous labor increased as the gestational age at admission increased, the difference between the rate of spontaneous labor of 67% at 28 to 32 weeks and 86% at 34 to 36 weeks was statistically significant (p = 0.001). The most common complication was clinical chorioamnionitis (6%) and postpartum sepsis (6%). The perinatal outcome was favorable in the majority of cases. Outcome improved with the increase in gestational age at PPROM. The overall perinatal mortality was 9.3%.[44]

MATERIALS AND METHODS

This was a prospective clinical study which was conducted in the department of Obstetrics and Gynaecology at Dayanand Medical College & Hospital Ludhiana (Punjab) over a period of 2 years, from September 2019 to September 2021.

SOURCE OF DATA

Patients admitted with preterm premature rupture of membranes between 24- 36 weeks 6days of gestation at Dayanand Medical College and Hospital, Ludhiana.

INCLUSION CRITERIA:

1. Women with a singleton pregnancy.
2. Gestation age between 24weeks-36 weeks 6days.
3. Women with preterm premature rupture of membranes.

EXCLUSION CRITERIA:

1. Multiple pregnancies
2. Fetal growth restriction
3. Uterine anomalies
4. Intrauterine fetal demise
5. Fetal anomalies
6. Any space-occupying lesion in the uterus
7. Hypertensive disorders and pregnancy-induced hypertension
8. Gestational diabetes mellitus
9. Chronic renal failure
10. Class II to IV cardiac diseases

STUDY SAMPLE:

The study was performed on 105 pregnant women admitted to labour ward of DMCH with PPROM. All women were between 24weeks – 36weeks 6 days of gestation. Out of 105 PPROM patients, 5 were lost to follow-up. Rest 100 women were followed up till delivery for the neonatal outcome.

METHODOLOGY:

Written and informed consent was taken from all the subjects entering the study. A detailed history was taken from all patients. A complete general, physical, systemic and obstetric examination was done initially to confirm the diagnosis of PPROM and to assess maternal & fetal condition. Signs of overt chorioamnionitis, such as foul-smelling discharge, tachycardia and fever were looked for. A per speculum examination was performed to confirm PPROM and to assess the cervical parameters. A high vaginal swab was taken for culture and sensitivity before initiation of antibiotics. No per vaginum examination was done unless the woman was in labour. A baseline obstetric ultrasound was done to assess fetal growth parameters, placenta and amniotic fluid volume. Women were observed in the labour room for at least 24 hours. Those not in labour and planned for conservative treatment were transferred to the high-risk maternal ward.

On admission, a non-stress test was done for all patients.

After confirming that all antenatal tests have been done, some specific tests were sent for PPROM that is, haemogram, C-reactive protein, urine routine, urine culture sensitivity and high vaginal swab. During the hospital stay, antenatal assessment including daily vital monitoring, obstetric examination, daily vulval pad estimation for continuing leaking, fetal growth assessment and estimation of liquor volume was done. Treatment was individualized and patients were managed as per standard protocols.

Data was made of the patients who required induction for obstetric indications. Mode of delivery was noted for all patients. Fetal parameters recorded were birth weight, Apgar score at 1 and 5 minutes, the necessity of NICU admission and respiratory complications mainly respiratory distress syndrome, transient tachypnea of newborn and congenital pneumonia.

We excluded meconium aspiration syndrome and perinatal asphyxia, from our study as they are pathophysiologically different from lung maturation.

Statistical analysis

Data were described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. For comparing categorical data, Chi square ($\chi 2$) test was performed and the exact test was used when the expected frequency is less than 5. A probability value (*p* value)less than 0.05 was considered statistically significant. All statistical calculations were done using (Statistical Package for the Social Science) SPSS 21 version (SPSS Inc., Chicago, IL, USA)statistical program for Microsoft Windows

OBSERVATION ANDRESULTS

In our study, a total of 105 pregnant women with preterm premature rupture of membranes between 24 weeks to 36 weeks 6days period of gestation were enrolled. Out of 105 PPROM patients, 5 were lost to follow up. Demographic, maternal and fetal parameters were recorded.

Demographic parameters

The following demographic parameters were noted:

1. Age
2. Education
3. Socio-economic class
4. Antenatal care
5. Gravidity

1. **Age (in years)**

TABLE 1: Distribution of Women under study according to age

Age group(yr)	No. of Cases (n=100)	Percentage
<25	15	15.00%
25-30	38	38.00%
31-35	40	40.00%
36-40	7	7.00%

In our study, PPROM was noted in 15 % of women in the age <25 years, 38 % women in the age group of 25-30 years, majority of women that is, 40% women were in the age group of 31-35 years, and 7% women between 36-40 years of age.

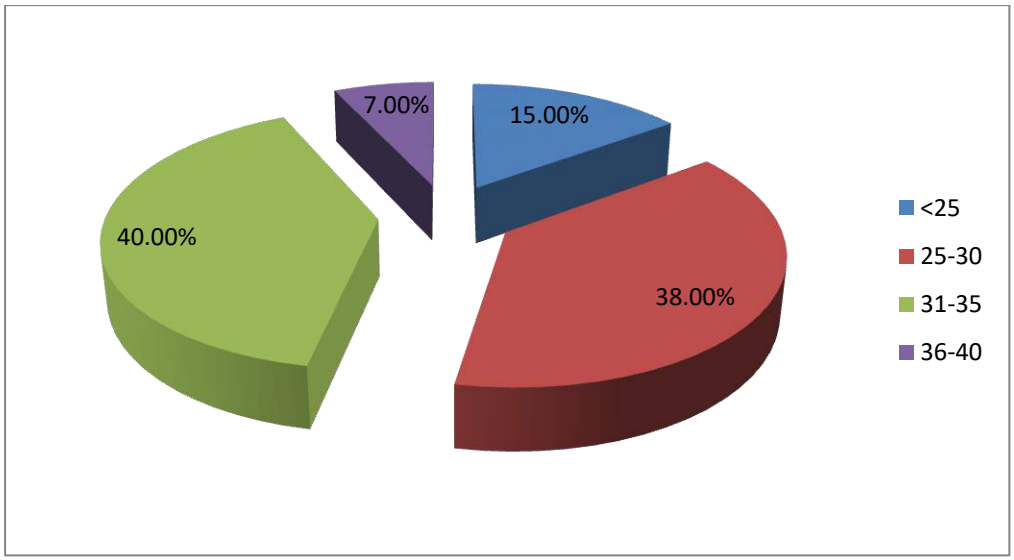


FIGURE 1: Distribution of women under study according to age

TABLE 2: Mean age distribution of women under study

	Total	Minimum	Maximum	Mean	Std. Deviation
Age	100	19.00	39.00	29.6200	4.43307

In our study, the mean age of the women presenting with PPROM was 29years +- 4.43 (19-39) years.

2. Education status

TABLE 3: Distribution of women under study according to education status

Education status	No of cases(n=100)	Percentage
Post graduate	20	20.0%
Graduate	52	52.0%
Others	28	28.0%

In our study, 52% women were graduates and 20% were post graduate. The rest 28% were either illiterate or had primary or secondary level education.

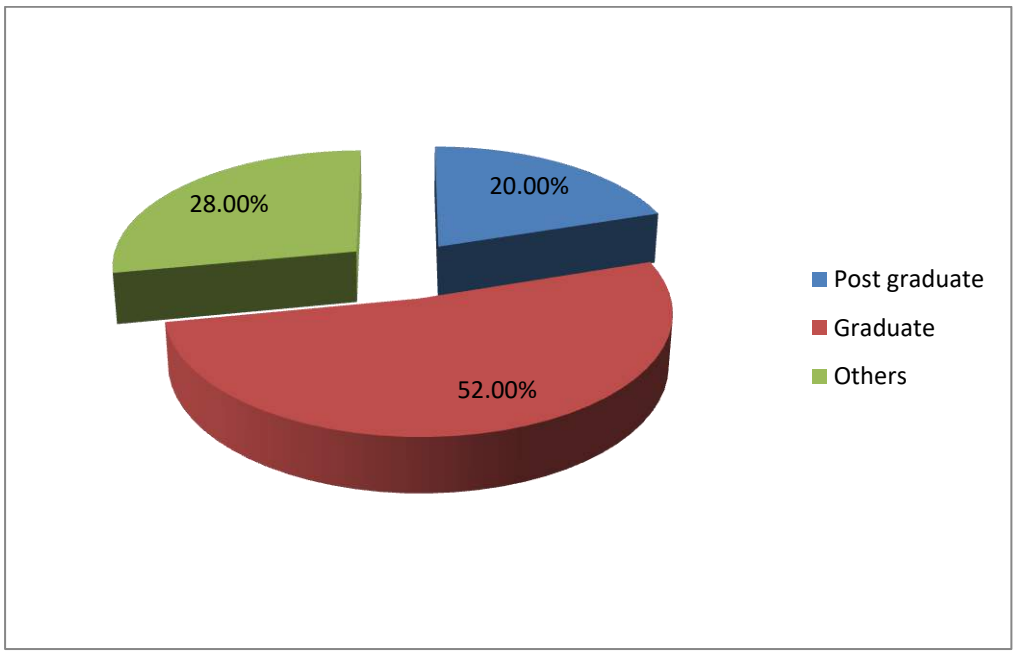


FIGURE 2: Distribution of women under study according to education status

3. Socio-economic class

Women were classified into the following 5 groups according to the modified kuppuswamy scale:

- Upper class(score 26-29)
- Upper middle class(score 16-25)
- Lower middle class (score 11-15)
- Upper lower class (score 5-10)
- Lower class (score below 5)

TABLE 4: Distribution of women under study according to socio-economic class

Socioeconomic class	No of cases (n=100)	Percentage
Upper class(26-29)	20	20.0%
Upper middle class(16-25	24	24.0%
Lower middle(11-15	34	34.0%
Upper lower class(5-10	15	15.0%
Lower class(<5)	7	7.0%

The majority of women that is, 34% were from a lower middle class family. 15% and 7% belonged to upper lower class and lower class respectively while 20% women belonged to upper class and 24% belonged to upper middle class.

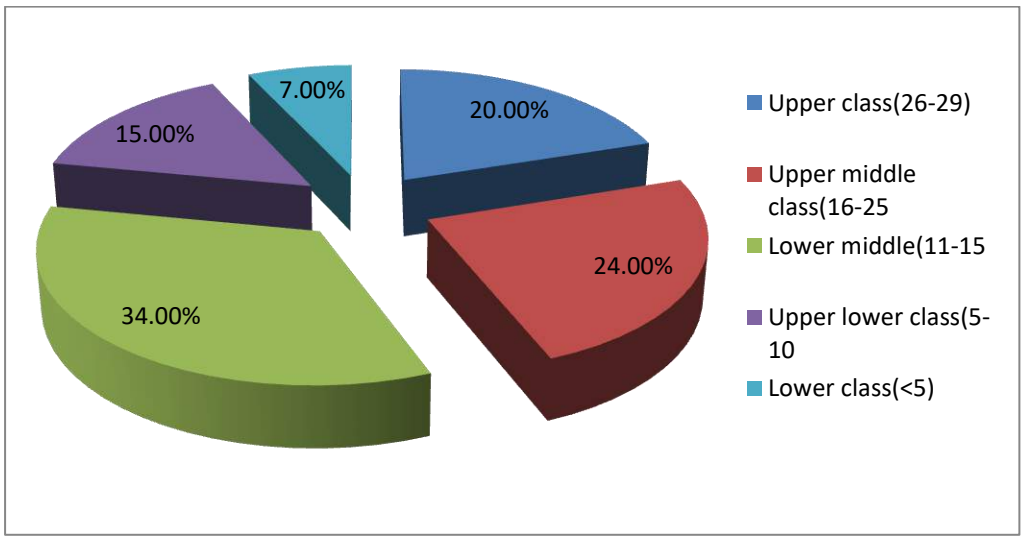


FIGURE 3: Distribution of women under study according to socio-economic class

4. Antenatal care

TABLE 5: Distribution of women under study according to antenatal care

Booked/unbooked	No of cases (n=100)	Percentage
Booked	91	91.0%
Unbooked	9	9.0%

In our study, 91% of pregnancies were booked cases and only 9% were unbooked. According to WHO, cases are considered booked if the pregnant lady has had a minimum of three visits for antenatal checkups after she was registered and confirmed to be pregnant.

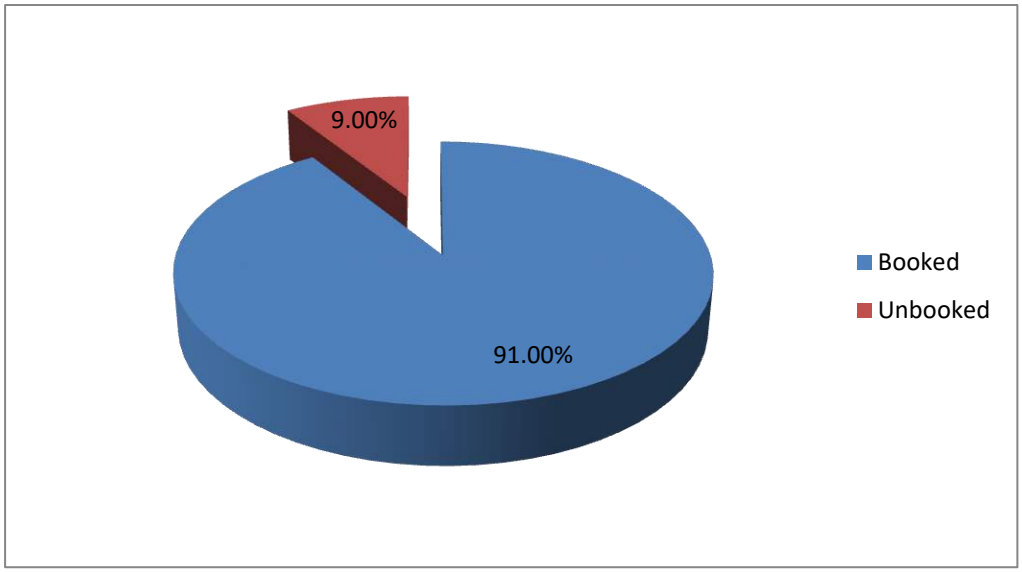


FIGURE 4: Distribution of women under study according to antenatal care

5. Gravidity

TABLE 6: Distribution of women under study according to gravidity

Parity	No of cases(n=100)	Percentage
Primigravida	52	52.0%
Multigravida	48	48.0%

In our study, 52% women presenting with PPROM were primigravida and 48% were multigravida.

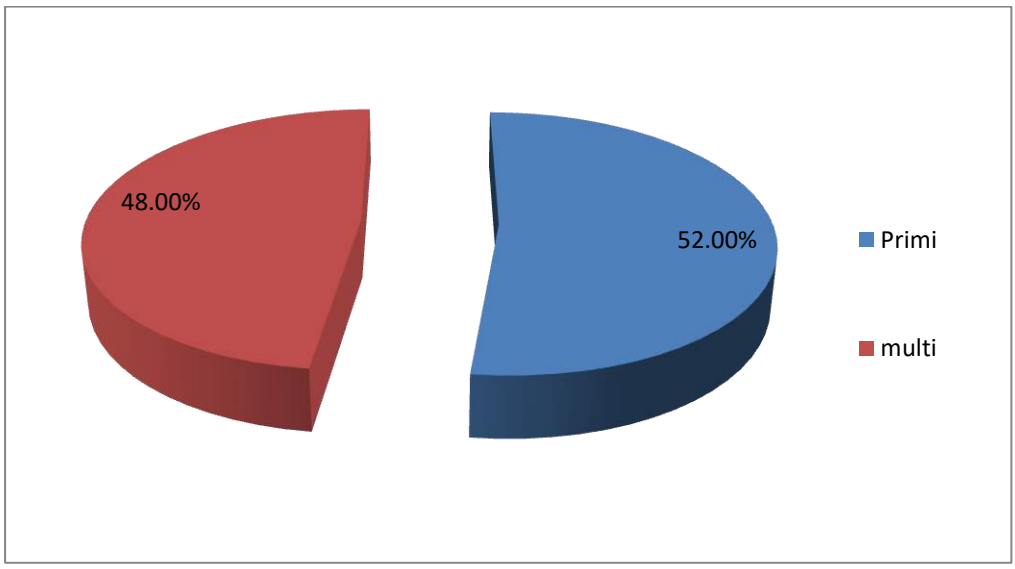


FIGURE 5: Distribution of women under study according to gravidity

Maternal parameters

The following maternal parameters were studied for all groups:

1. Gestational age
2. Risk factors
3. Duration of leaking
4. Colour of liquor
5. Duration of labour pains with pprom
6. Association of UTI
7. Association of genital infection
8. Amniotic Fluid Index (AFI)
9. Antenatal steroid cover
10. Induction of labour

1. Gestational age (in weeks)

Out of 100 women in our study, 12 women with PPROM had a gestation of 24weeks to 28weeks at admission and this formed Group A. Group B comprised of 55 women between gestational age of 28weeks1day and 33 weeks 6 days and Group C comprised of 33 women between 34weeks to 36 weeks 6days period of gestation.

- Group A :extremely preterm- 24weeks-28weeks(n=12)
- Group B :early preterm- 28^{+1}weeks – 33^{+6}weeks (n=55)
- Group C:late preterm- 34weeks- 36^{+6}weeks (n=33)

The mean gestational age at presentation was 32.2+- 3weeks with majority of women (55%) between 28weeks 1day and 33weeks 6days period of gestation.

COMPARISON OF VARIOUS DEMOGRAPHIC AND MATERNAL PARAMETERS OF GROUP-A, B AND C.

TABLE 7: Comparison of age of the three groups

Gestational age	<25years		25-30years		31-35years		36-40years		p-value
24-28 weeks(n=12)	4	33.33%	3	25.00%	3	25.00%	2	16.67%	0.101
28^{+1}-33^{+6} weeks(n=55)	8	14.55%	23	41.82%	20	36.36%	4	7.27%	0.824
34-36^{+6} weeks(n=33)	3	9.09%	12	36.36%	17	51.52%	1	3.03%	0.001
Total(n=100)	15	15.00%	38	38.00%	40	40.00%	7	7.00%	

Chi-square value-8.534; p-value-0.202

It was seen that among patients less than 25years (33.33%) and patients more than 36 years (16.67%) majority of cases of PPROM occurred at extreme preterm gestational age. PPROM was seen at relatively later gestational ages between the age group of 25-35 years. This difference in age at presentation of PPROM in the late preterm group was statistically significant (p-value 0.001).

Overall, there was statistically no significant difference among the three groups (p value 0.2).

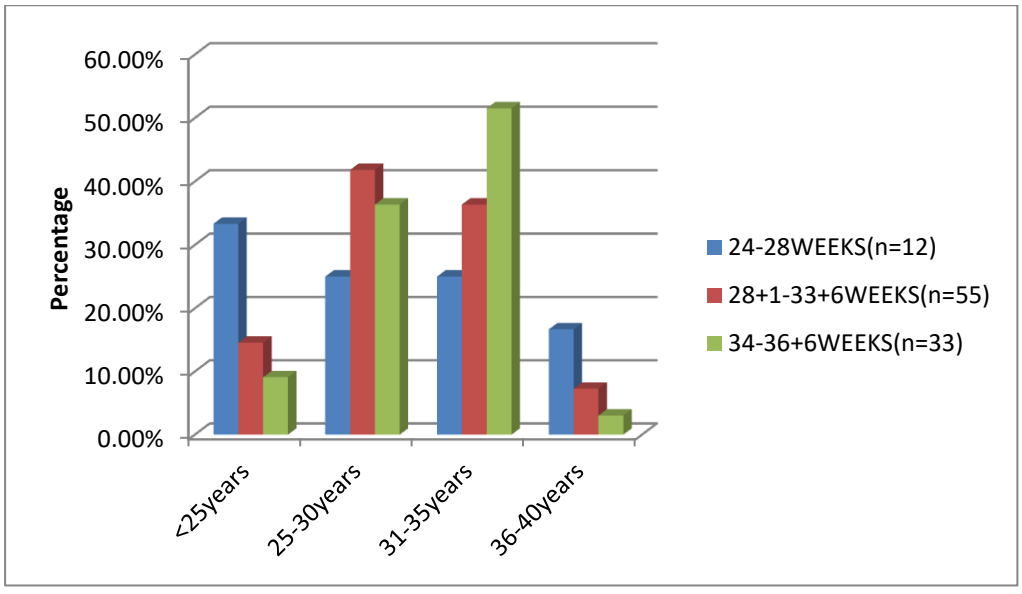


FIGURE 6: Comparison of age of the three groups

TABLE 8: Comparison of education levels of the three groups

Gestational age	Post graduate		Graduate		Others		p-value
24-28 weeks(n=12)	1	8.33%	3	25.00%	8	66.67%	0.048
28^{+1}-33^{+6} weeks(n=55)	9	16.36%	31	56.36%	15	27.27%	0.040
34-36^{+6} weeks(n=33)	10	30.30%	11	33.33%	12	36.36%	0.126
Total (n=100)	20	20.00%	45	45.00%	35	35.00%	

Chi-square value-10.968; p-value-0.026

Out of 20 postgraduate women, 30% were in the late preterm gestational age when they presented with leaking per vaginum and only 8% belonged to extreme preterm group. When compared among the patients having gestation less than 28weeks at presentation of PPROM, it was seen that percentage of patients increased as the education level decreased. The difference was found to be statistically significant (p-value- 0.04)

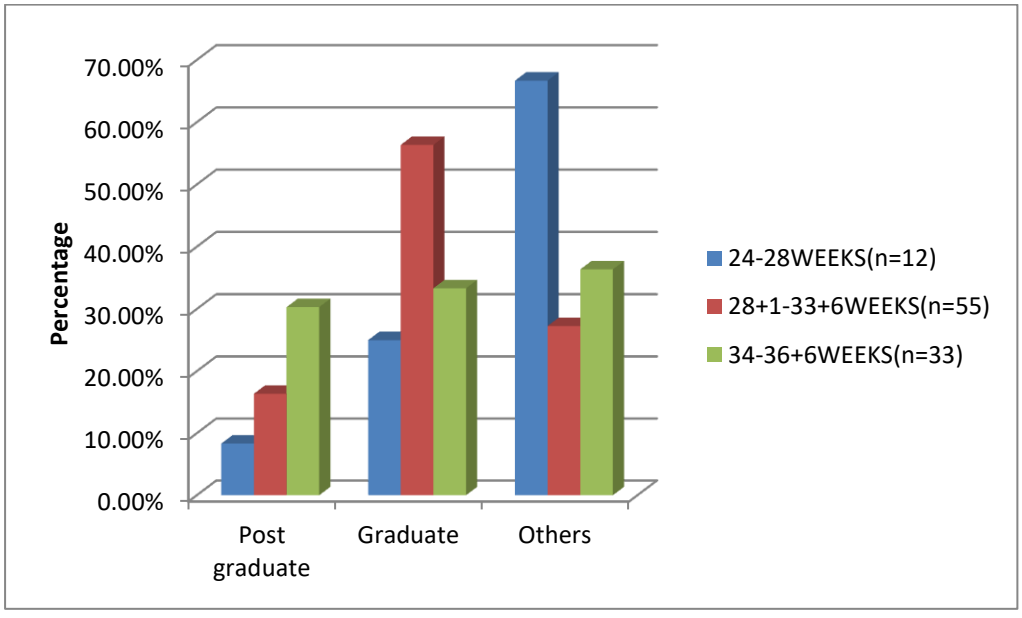


FIGURE 7: Comparison of education levels of the three groups

TABLE 9: Comparison of socio-economic status of the three groups

Gestational age	Upper class		Upper middle		Lower middle		Upper lower		Lower		p-value
24-28 weeks(n=12)	1	8.33%	2	16.67%	2	16.67%	4	33.33%	3	25.00%	0.018
28^{+1}-33^{+6} weeks(n=55)	7	12.73%	13	23.64%	24	43.64%	8	14.55%	3	5.45%	0.135
34-36^{+6} weeks(n=33)	12	36.36%	9	27.27%	8	24.24%	3	9.09%	1	3.03%	0.033
Total (n=100)	20	20.00%	24	24.00%	34	34.00%	15	15.00%	7	7.00%	

Chi-square value-20.546; p-value-0.008

Majority of the patients between 34weeks to 36weeks6days period of gestation (36.36%) belonged to upper class. Most of the patients who presented with leaking at less than 28weeks gestation belonged to upper lower and lower class. The difference was found to be statistically significant in Group A and Group C (p-value 0.018 and 0.033 respectively) concerning socio-economic class. Overall, the difference in socio economic class and gestational age was found to be statistically significant in 3 groups (p value 0.008).

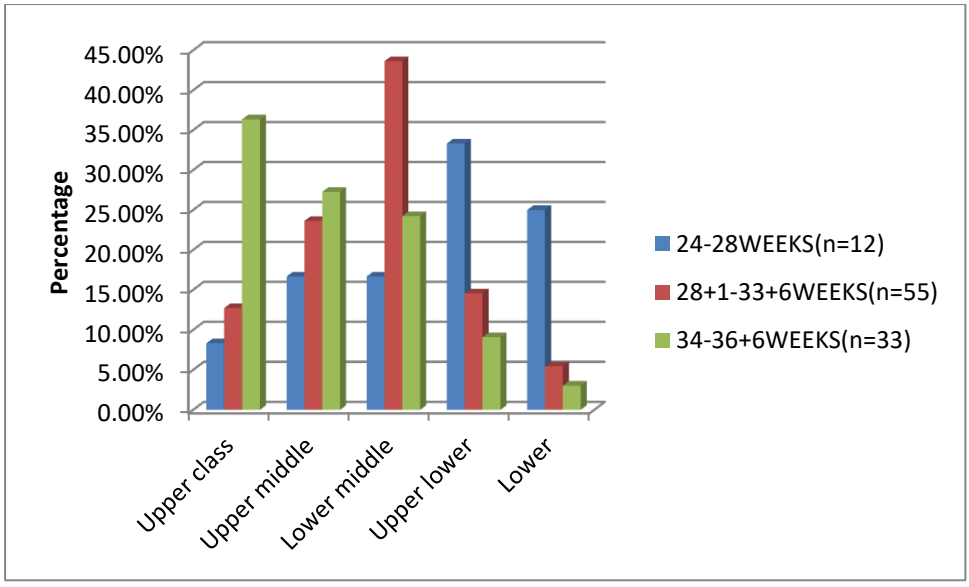


FIGURE 8: Comparison of socio-economic status of the three groups

TABLE 10: Comparison of mode of delivery in various groups

Gestational age	Vaginal delivery		LSCS		Instrumental delivery		p-value
24-28 weeks(n=12)	8	66.67%	4	33.33%	0	0.00%	0.315
28^{+1}-33^{+6} weeks(n=55)	29	52.73%	26	47.27%	0	0.00%	0.288
34-36^{+6} weeks(n=33)	10	30.30%	20	60.61%	3	9.09%	0.020
Total (n=100)	47	47.00%	50	50.00%	3	3.00%	

Chi-square value-7.213; p-value-0.125

In our study, 47 patients presenting with PPROM delivered vaginally , 3 underwent instrumental delivery (forceps) and the rest 50 underwent cesarean section. It was seen that in Group A , patients delivered mostly vaginally (66.67%). In Group B, the percentage of patients undergoing cesarean section and normal delivery was almost comparable. In Group C, 60% patients underwent cesarean section i.e. the rate of cesarean section increased at late preterm gestation.3 patients from this group underwent forceps delivery. The difference in mode of delivery was statistically significant (p-value 0.02) in this group.

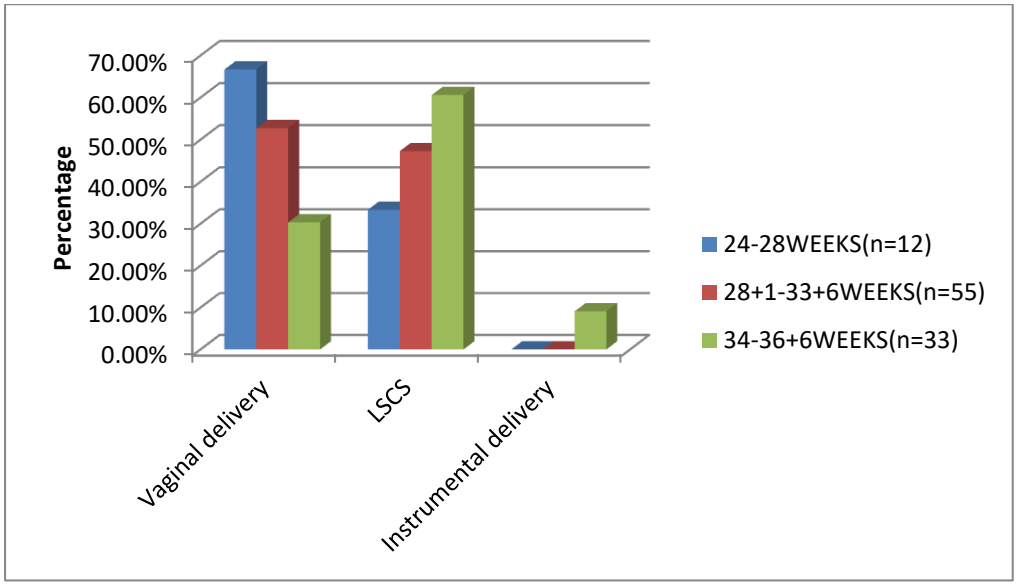


FIGURE 9: Comparison of mode of delivery in various groups

2. Risk factors for PPROM

TABLE 11: Distribution of women under study according to risk factors for PPROM

Risk Factors	Cases (n=100)	Percentage
Environmental	7	7.0%
Lifestyle(smoking, drug)	2	2.0%
Excess/ Low Maternal weight gain	17	17.0%
Psychological (depression, anxiety)	1	1.0%
Increased physical activity	12	12.0%
Genetic	10	10.0%
Bad Oro dental hygiene	2	2.0%
Interval b/w pregnancies <18mnth	15	15.0%
Prior preterm birth	30	30.0%
Infection	32	32.0%

*b/w: between

In our study, the most commonly encountered risk factor in women with PPROM was genitourinary infection which was present in 32% patients. The second most common risk factor was a history of prior preterm birth (30%). Multiple risk factors were also seen in a single patient. The least common risk factors seen in only 2% patients were bad orodental hygiene and faulty lifestyle habits like consumption of drugs and smoking.

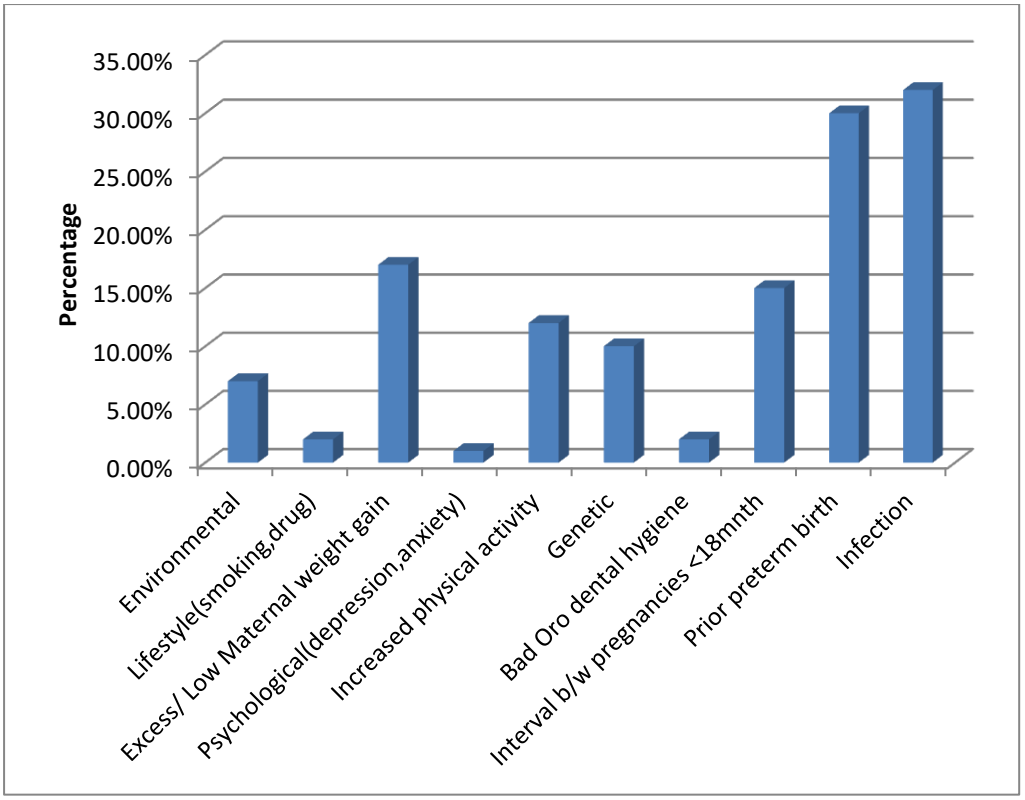


FIGURE 10: Distribution of women under study according to risk factors for PPROM

3. Duration of PPROM

TABLE 12: Distribution of women under study according to duration of PPROM

Leaking per vaginum (time in hrs)	No of cases	Percentage
<24	58	58.0%
>24	42	42.0%

In our study, 58% of the total patients presented with preterm premature rupture of membranes of duration less than 24hours and 42% had a history of leaking of more than 24hours.

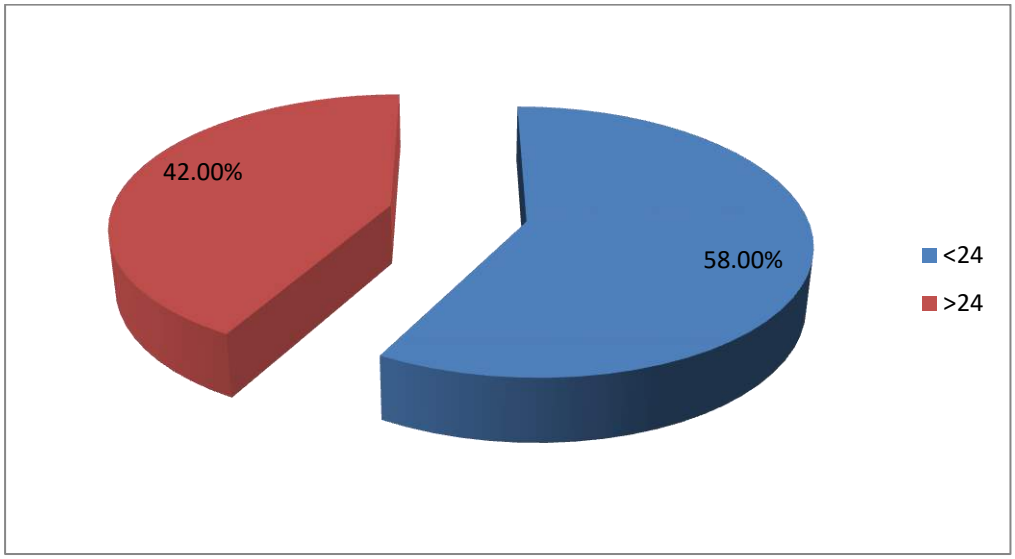


FIGURE 11: Distribution of women under study according to duration of PPROM

TABLE 13: Mean duration of PPROM at the time of admission

	N	Minimum	Maximum	Mean	Std. Deviation
2. h/o Leaking since (Hours)	100	1.00	72.00	22.1400	19.47545

The mean duration of leaking at the time of admission to the hospital was 22hours with a range between 1 hour and 72 hours.

4. Colour of liquor

TABLE 14: Distribution of women under study according to colour of liquor

Color of liquor	Cases(n=100)	Percentage
Clear	80	80.0%
Blood tinged	8	8.0%
Meconium stained	12	12.0%

On examination it was seen that in 80% patients , the liquor was clear, watery in consistency and non foul smelling. In 12% patients it was stained with meconium and in the rest 8%, it was blood tinged.

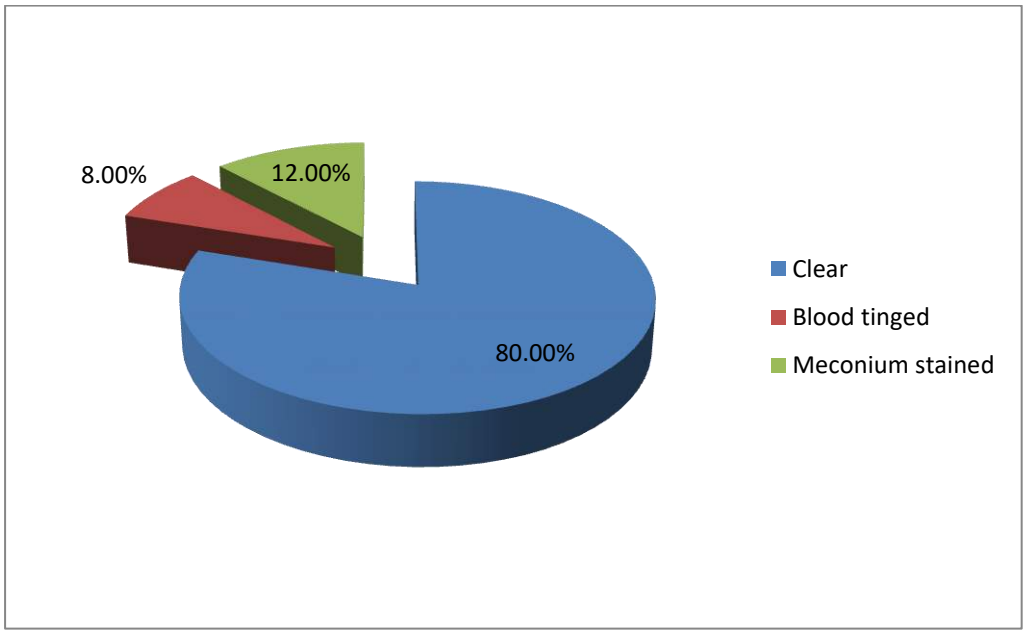


FIGURE 12: Distribution of women under study according to colour of liquor

5. Duration of labour pains with PPROM

TABLE 15: Distribution of women under study according to duration of labour pains

Labour pains with PPROM	No of cases(n=55)	Percentage
<24hrs	25	45.5%
>24hrs	30	54.5%

In our study, 55 patients presented with labour pains along with preterm premature rupture of membranes. 45.5% patients had labour pains of duration less than 24 hours and 54.5% had associated labour pains of more than 24 hours duration.

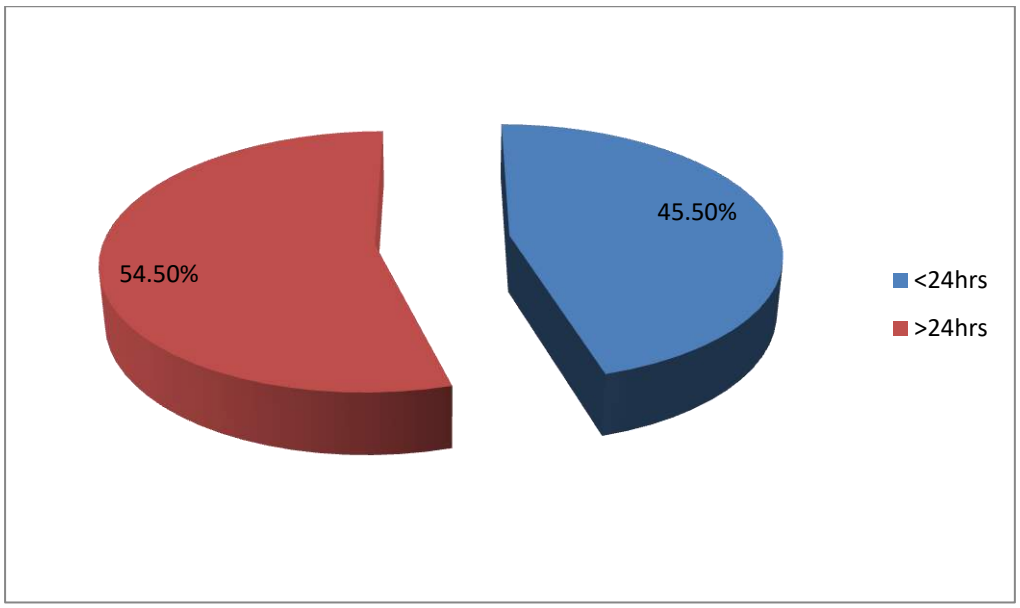


FIGURE 13: Distribution of women under study according to duration of labour pains

6. Association of urinary tract infection with PPROM

A) Urine routine examination

TABLE 16: Distribution of women under study according to urine routine results

Urine r/ examination (no. of pus cells)	Cases (n=100)	Percentage
0-5	26	26.0%
>5	52	52.0%
Others	22	22.0%

Urine routine examination was done in all patients. Patients were divided into 3 groups based on the presence of pus cells or other components like yeast or calcium oxalate crystals .26% patients had an insignificant number of pus cells (0-5), 52% had more than 5 pus cells and the rest 22% had other components.

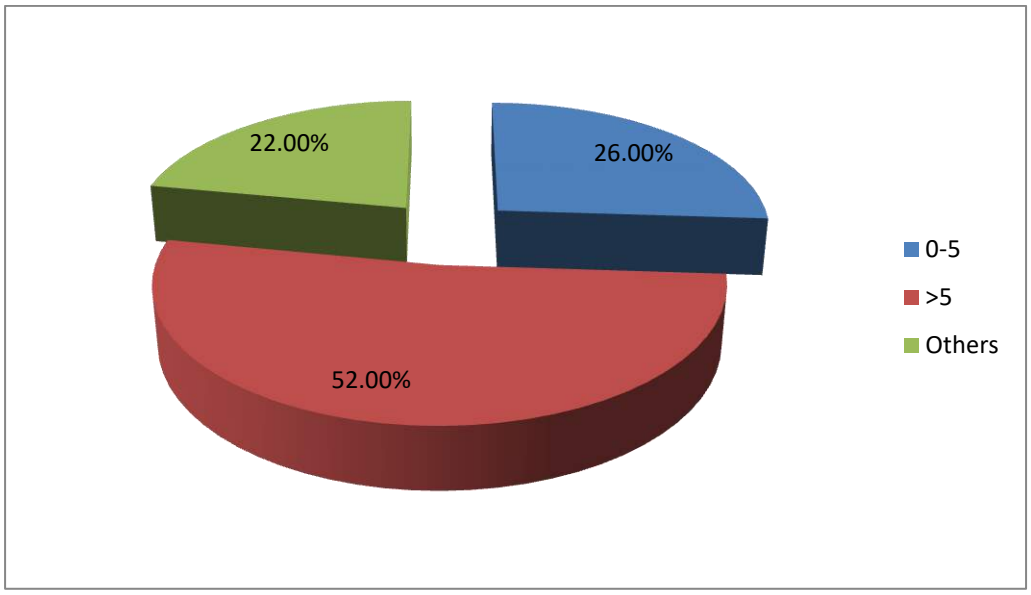


FIGURE 14: Distribution of women under study according to urine routine results

B) Urine culture & sensitivity

TABLE 17: Distribution of women under study according to urine C/S report

Urine culture	No of cases(n=100)	Percentage
No growth	40	40.0%
Significant Growth present	54	54.0%
Contamination	6	6.0%

Urine culture and sensitivity testing was done in all patients. 40% patients had no growth, 54% had significant growth and 6% samples were contaminated.

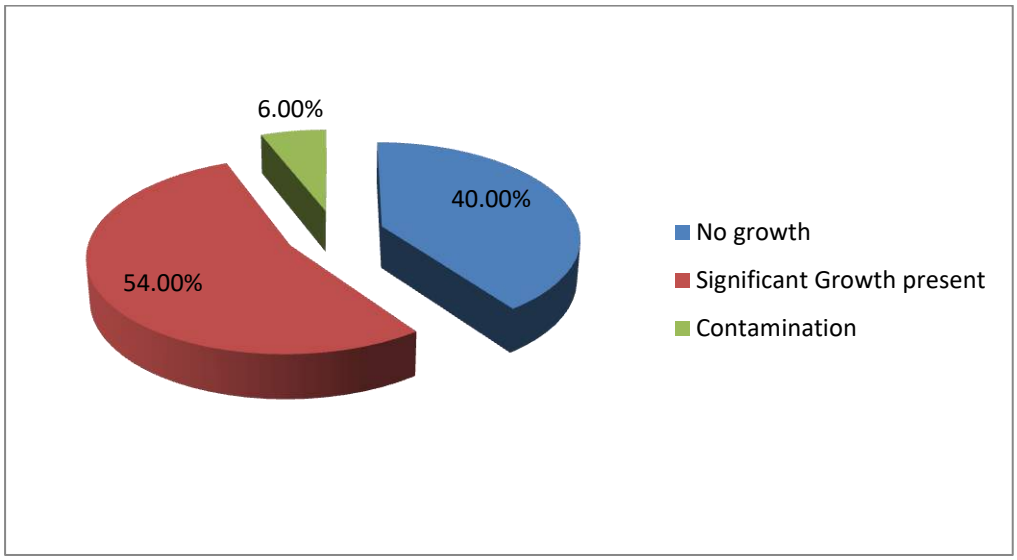


FIGURE 15: Distribution of women under study according to urine C/S report

*C/S= Culture & Sensitivity

C) Causative organism

TABLE 18: Distribution of women according to causative organism of UTI

Urine C/S growth type	No of cases(n=54)	Percentage
E. coli	22	40.7%
Enterobacter cloacae	1	1.9%
Klebsiela	16	29.6%
Enterococcus faecalis	2	3.7%
Proteus species	3	5.6%
Pseudomonas aeruginosa	10	18.5%

Out of the 54 samples with significant growth, it was seen that E.coli was present predominantly in 40.7% samples followed by Klebsiella (29.6%). The least common organism found in only 1 case was Enterobacter cloacae.

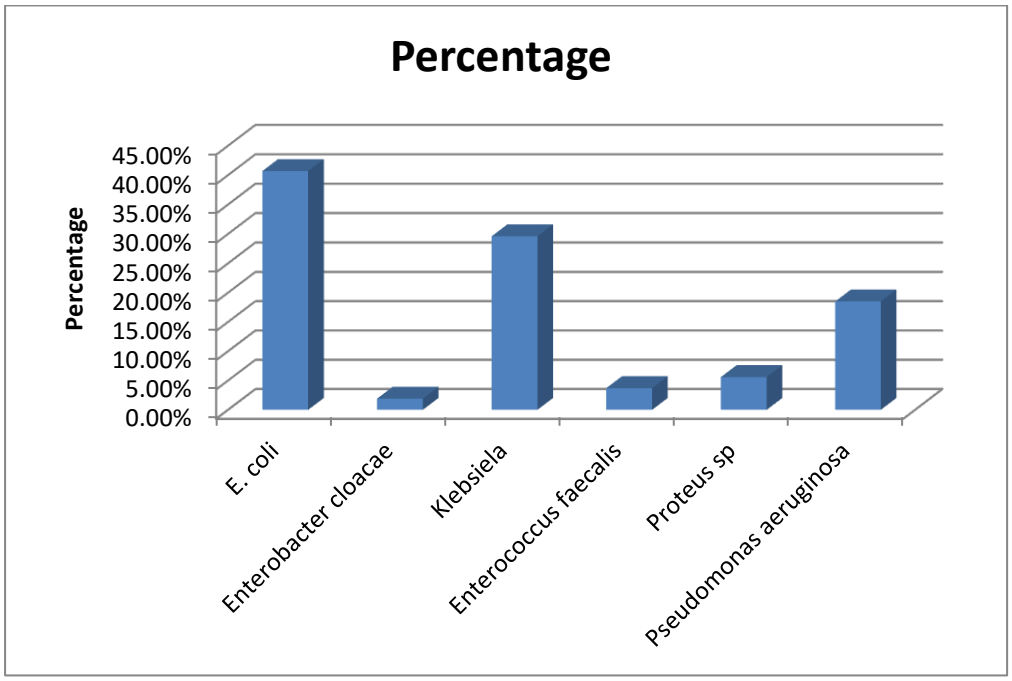


FIGURE 16: Distribution of women according to causative organism of UTI

7. Association of genital infection with PPROM

A) High vaginal swab

TABLE 19: Distribution of women according to high vaginal swab culture report

High vaginal swab c/s	No. of cases(n=100)	Percentage
No growth	38	38.0%
Significant Growth present	60	60.0%
Contamination	2	2.0%

All patients were subjected to a per speculum examination and high a vaginal swab was taken. Significant growth was present in 60 patients while 38 patients had no growth.

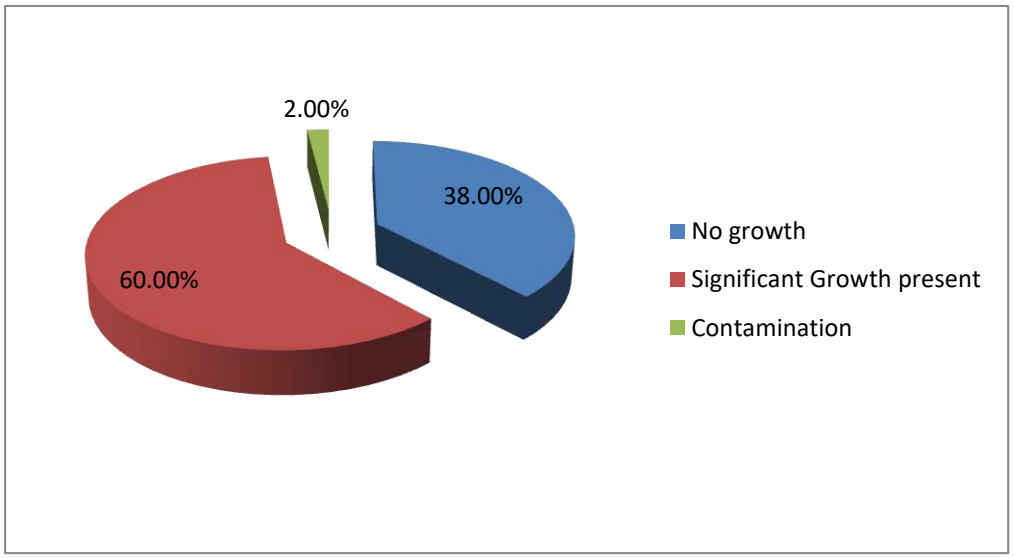


FIGURE 17: Distribution of women according to high vaginal swab culture report

B) Causative organism

TABLE 20: Distribution of women according to causative organism for genital infection

HVS growth type	No of cases(n=60)	Percentage
Candida	15	25.0%
E. coli	30	50.0%
Klebsiela	8	13.3%
Enterococcus faecium	3	5.0%
Staph. aureus	2	3.3%
Mixed growth	2	3.3%

On culture and sensitivity testing, it was seen that 50% patients had a culture report suggestive of E.coli. It was followed by Candida infection in 25% patients while only 3.3% had reports suggesting Staphylococcus aureus and mixed growth.

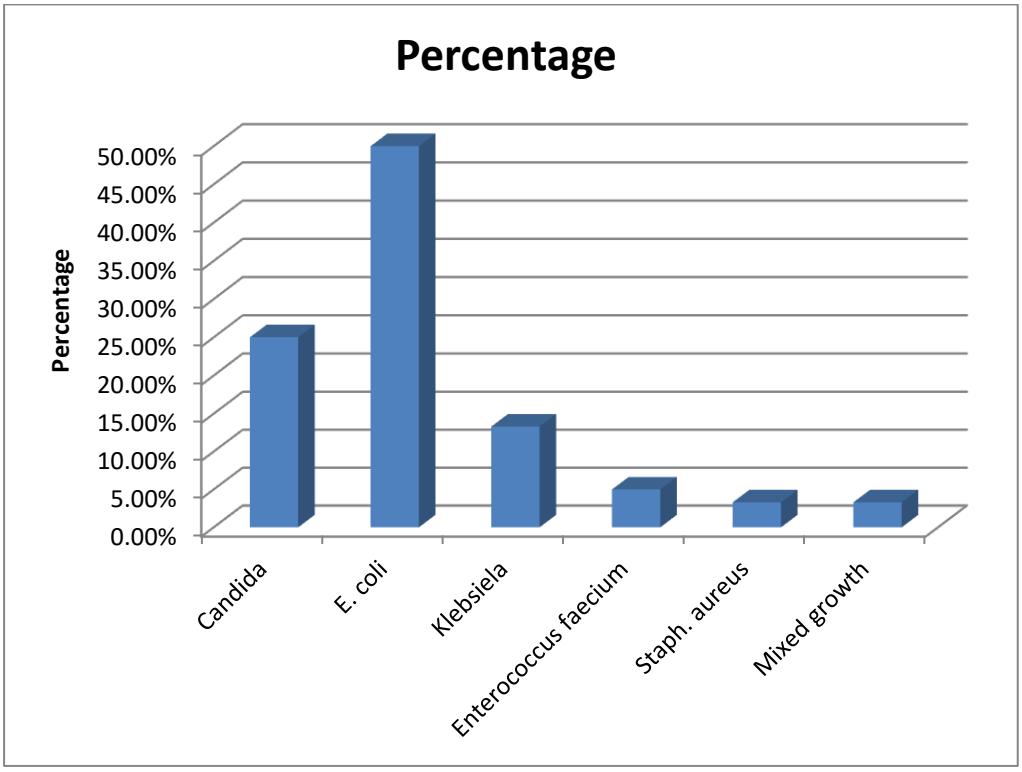


FIGURE 18: Distribution of women according to causative organism for genital infection

8. Amniotic fluid index (AFI) at presentation of PPROM

TABLE 21: Distribution of women according to AFI at presentation

AFI(in cm)	Cases (n=100)	Percentage
<5	25	25.0%
5-8	42	42.0%
>8	33	33.0%

Amniotic fluid index (AFI)was checked for each patient . AFI < 5cm is severe oligohydramnios, 5-8 cm is mild oligohydramnios and >8 upto 18cm is normal. In our study, majority of the patients (42%) had mild oligohydramnios , 25% patients had severe oligohydramnios and 33% patients had normal AFI.

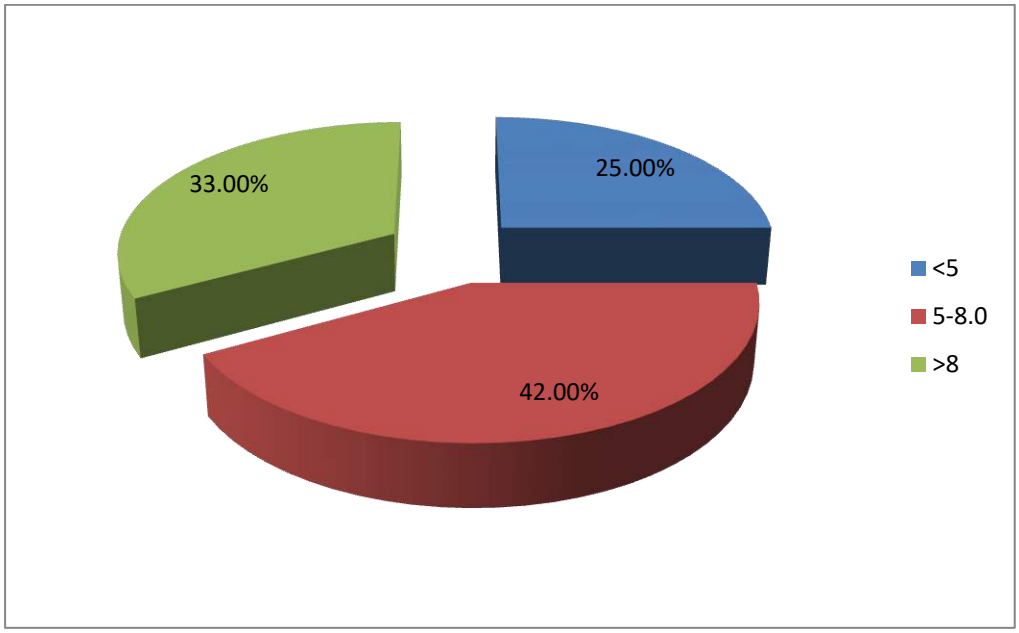


FIGURE 19: Distribution of women according to AFI at presentation

9. Antenatal steroid cover

TABLE 22: Distribution of women according to Antenatal steroid cover

Antenatal steroid cover	No of cases (n=100)	Percentage
Complete	54	54.0%
Incomplete	21	21.0%
Not received	25	25.0%
Rescue dose	0	0%

Out of the 100 patients in our study, 54% had complete antenatal corticosteroid cover before delivery. Twenty one patients could not receive the complete course as they delivered before the last dose could be given. After 34 weeks, antenatal corticosteroids were not given (n=25) according to standard protocols. No rescue dose of steroids was given.

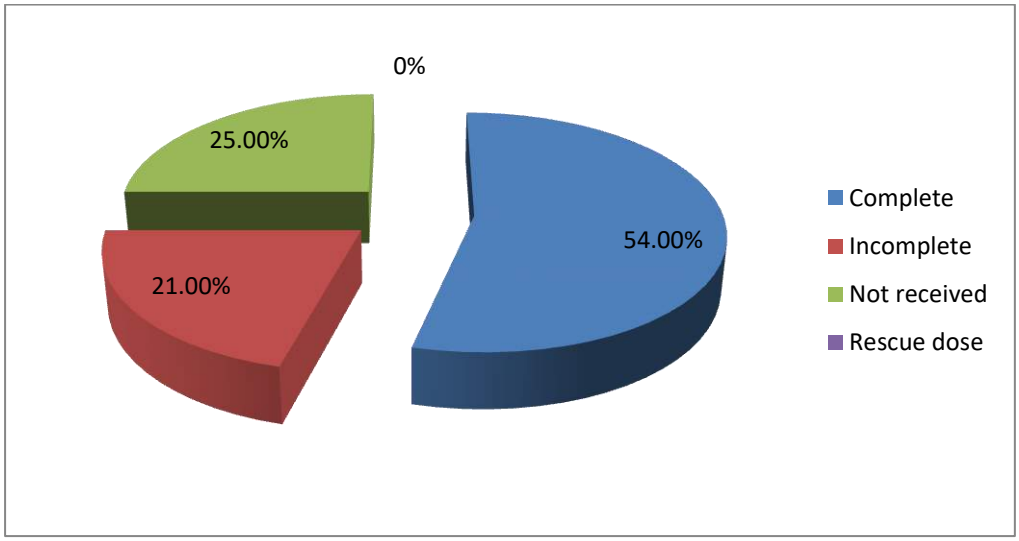


FIGURE 20: Distribution of women according to Antenatal steroid cover

10. Induction of labour for patients of PPROM

TABLE 23: Distribution of women according to induction of labour

Induction of labour	Cases (n=100)	Percentage
Induced	45	45.0%
Spontaneous labour	55	55.0%

Induction of labour was done in 45% of the patients while 55% patients went into spontaneous labour.

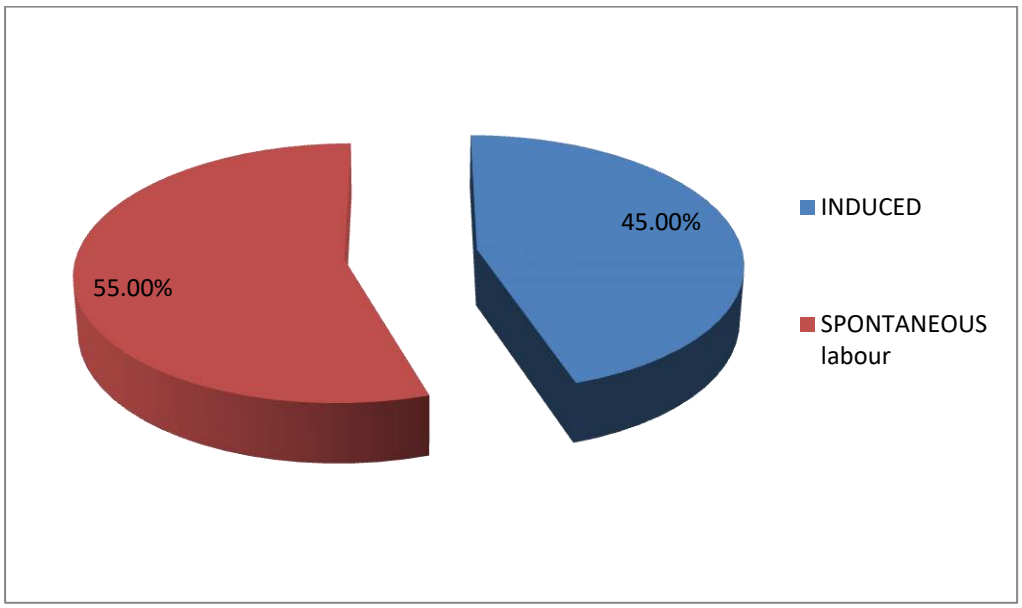


FIGURE 21: Distribution of women according to induction of labour

Fetal parameters

1. Birth weight
2. Apgar score
3. NICU admission
4. Respiratory complications

1. Birth weight

   Neonates were divided into 4 groups on the basis of birth weight

   - Birth weight > 2500 grams
   - LBW- low birth weight – weight at birth <2500grams
   - VLBW- very low birth weight – weight at birth <1500grams
   - ELBW- extremely low birth weight – weight at birth <1000grams

TABLE 24: Distribution of neonates according to birth weight

Birth weight	No. of babies(n=100)	Percentage
>2500gm	12	12.0%
(LBW) <2500gm	48	48.0%
(VLBW)<1500gm	29	29.0%
(ELBW)<1000gm	11	11.0%

Out of 100 babies born, 48% were low birth weight, 29% were very low birth weight and 11% were extremely low birth weight. Twelve babies weighed more than 2500 grams.

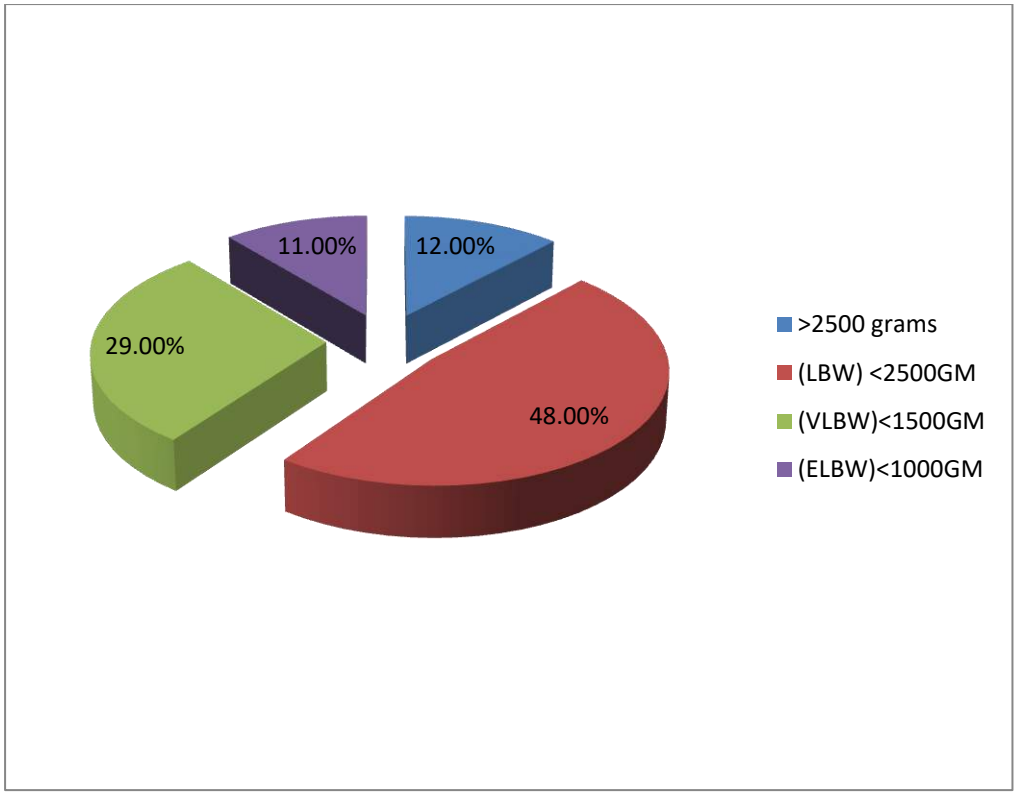


FIGURE 22: Distribution of neonates according to birth weight

2. Apgar Score

TABLE 25: Distribution of neonates according to Apgar score

Apgar score	<7		>=7	
AT 1 min (n=100)	66	66.0%	34	34.0%
AT 5 min (n=100)	56	56.0%	44	44.0%

Apgar score was measured at 1 and 5 minutes of birth for all newborn babies. 66% newborns had a score of <7 at 1minute and 34% newborns had a score >7. At 5 minutes, 56% of total newborns had an Apgar score <7 and 44% had a score of >7.

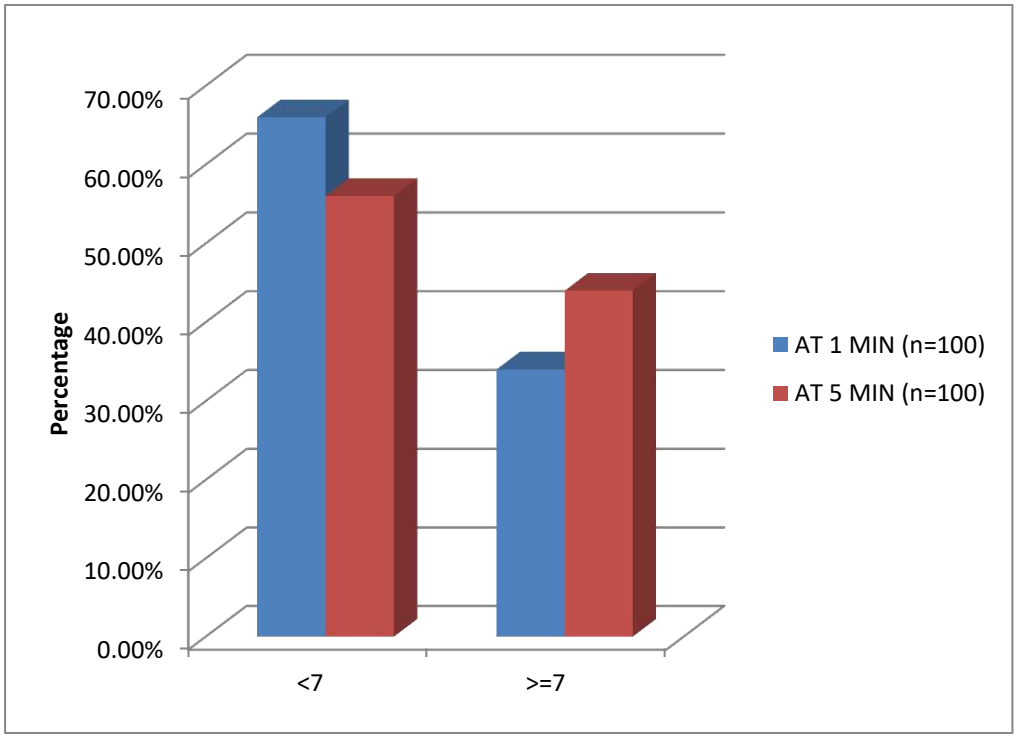


TABLE 23: Distribution of neonates according to Apgar score

COMPARISON OF APGAR SCORE AT 1 AND 5 MINUTE OF THE THREE GROUPS

TABLE 26: Comparison of Apgar score at 1 min of the three groups

Gestational age	<7		>=7		p-value
24-28 weeks(n=12)	12	100.0%	0	0.0%	0.001
28^{+1}-33^{+6} weeks(n=55)	46	83.6%	9	16.4%	0.001
34-36^{+6} weeks(n=33)	8	24.2%	25	75.8%	0.001
Total (n=100)	66	66%	34	34%	

It was seen that all extreme preterm babies (100%) had an Apgar score of less than 7. In Group B, 83.6% newborns had a score of <7 and 16.4% had a score of more than or equal to 7. In late preterm group, 3/4th of the newborns had an Apgar score of >7. The difference in Apgar scores at 1 min was statistically significant (p-value 0.001) among the three gestational groups.

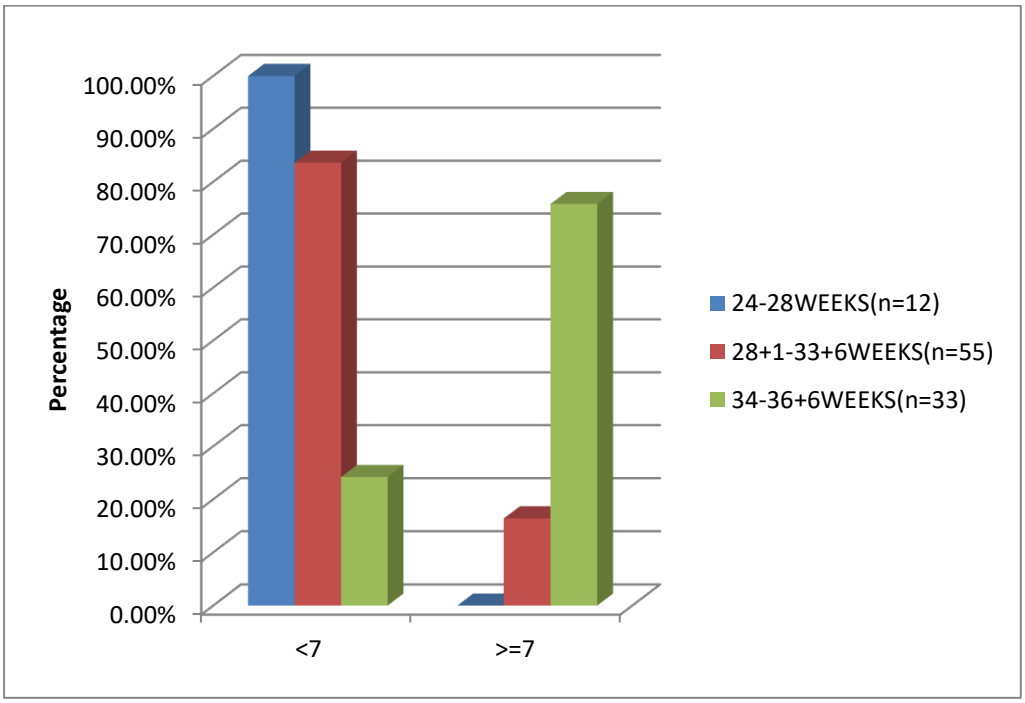


FIGURE 24: Comparison of Apgar score at 1 min of the three groups

TABLE 27: Comparison of Apgar score at 5 min of the three groups

Gestational age	<7		>=7		p-value
24-28 weeks(n=12)	12	100.0%	0	0.0%	0.001
28^{+1}-33^{+6} weeks(n=55)	31	56.4%	24	43.6%	0.001
34-36^{+6} weeks(n=33)	3	9.0%	30	90.9%	0.001
Total (n=100)	46	46%	54	54%	

At 5 minutes of birth, Apgar score was monitored in all the newborns. All extreme preterms had an Apgar score of less than 7 even at 5 minutes. In group C, 90.9% of newborns had an Apgar score of >7 at 5 minutes. The difference in Apgar scores at 5 min was statistically significant (p-value 0.001) among the three gestational groups.

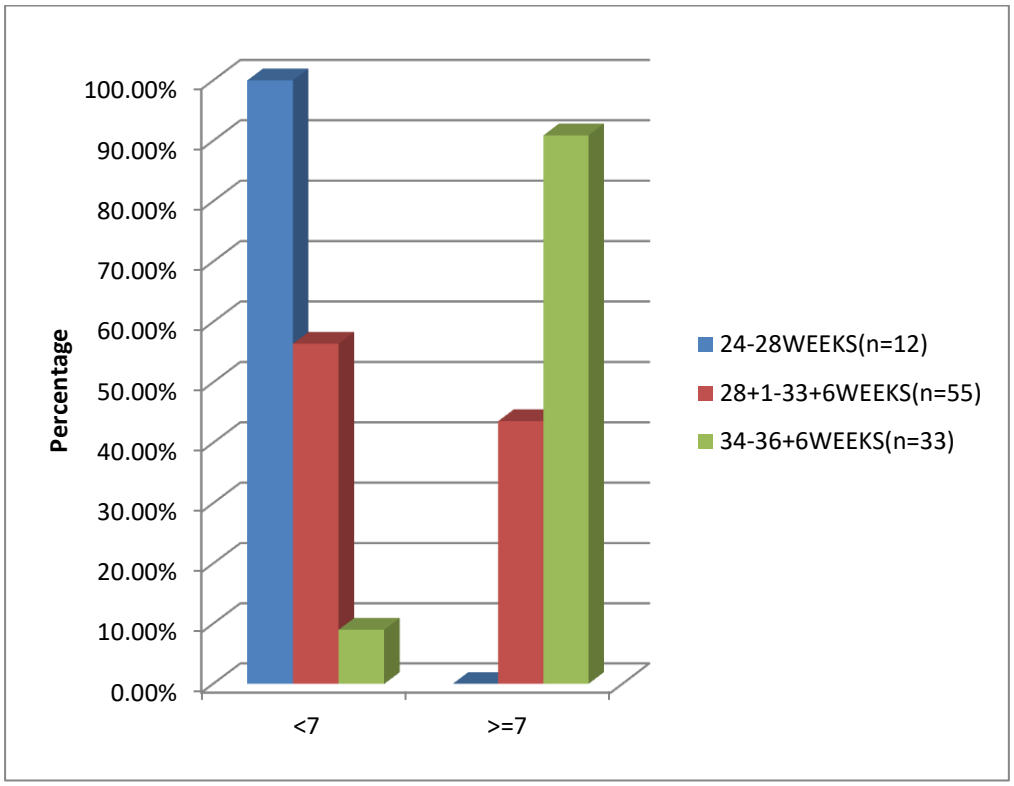


FIGURE 25: Comparison of Apgar score at 5 min of the three groups

3. NICU Admission

TABLE 28: Distribution of neonates according to NICU admission

Baby shifted to NICU	No. of cases(n=100)	Percentage
Yes	62	62.0%
No	38	38.0%

Out of 100 newborns, 62 were shifted to Neonatal ICU and the rest 38 neonates were either shifted to mother side or kept in observation room for a few hours and then shifted to mother.

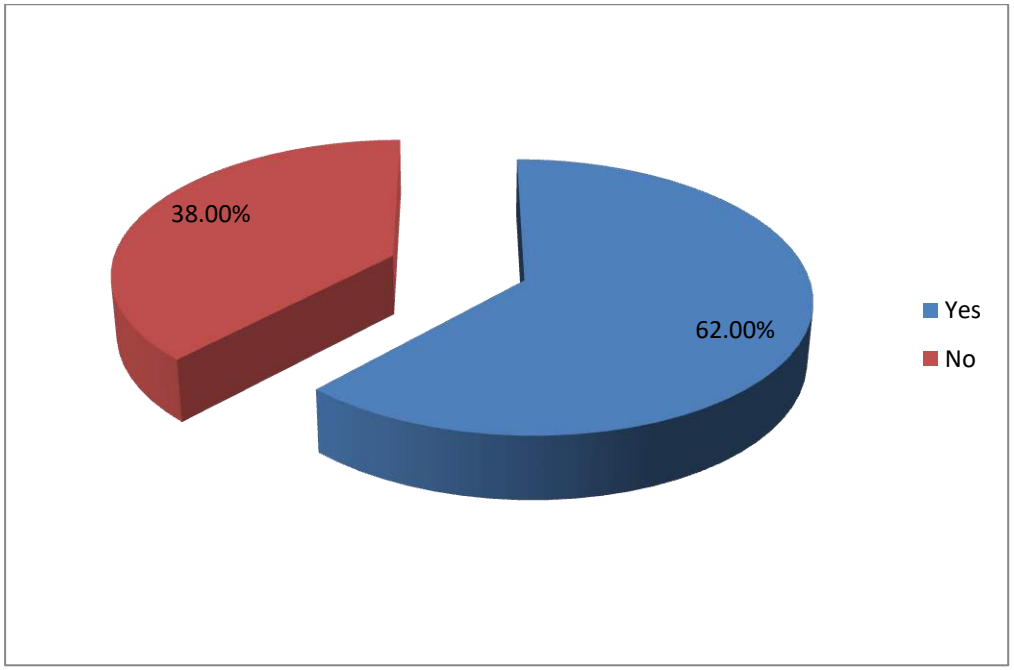


FIGURE 26: Distribution of neonates according to NICU admission

TABLE 29: Comparison of NICU admissions of the three groups

Gestational age	Shifted to NICU (n=62)	Percentage	p-value
24-28 weeks(n=12)	12	100%	0.0001
28^{+1}-33^{+6} weeks(n=55)	45	81.8%	
34-36^{+6} weeks(n=33)	5	15.1%	

All neonates (100%) born at <28weeks were shifted to Neonatal ICU while only 5 neonates (15%)in the period of gestation of 34-36weeks6days were shifted to NICU.From the early preterm group, NICU admissions were 81.8%. This difference in NICU admissions was statistically significant (p-value 0.0001).

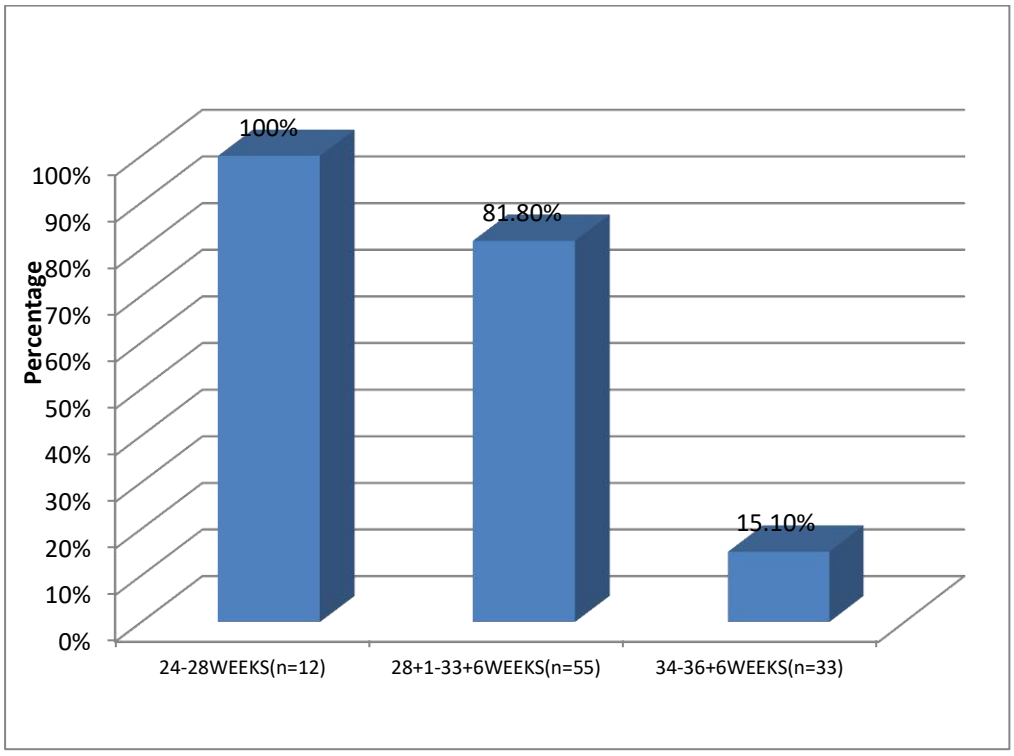


FIGURE 27: Comparison of NICU admissions of the three groups

4. Respiratory complications

TABLE 30: Distribution of neonates according to respiratory complications

Respiratory Complications	No. of cases(n=100)	Percentage
RDS	38	38.0%
TTNB	8	8.0%
Pneumonia	19	19.0%
None	35	35.0%

Three respiratory complications were followed up in babies of patients with PPROM in our study that is, Respiratory distress syndrome, Transient tachypnea of newborn and Congenital pneumonia. It was seen that respiratory distress was the most common respiratory complication amongst the three (38%). 35% neonates had no respiratory complications. Transient tachypnea of newborn was seen in 8% and congenital pneumonia was seen in 19% newborns.

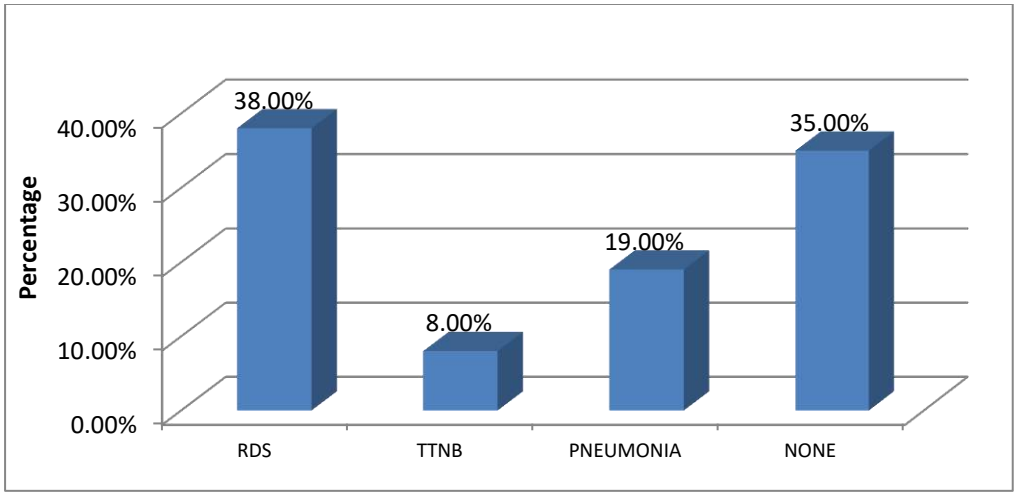


FIGURE 28: Distribution of neonates according to respiratory complications

COMPARISON OF RESPIRATORY COMPLICATIONS

TABLE 31: Comparison of respiratory distress syndrome in neonates of the three groups

Gestational age	Respiratory distress (n=38)	Percentage	p-value
24-28 weeks(n=12)	12	100.0%	0.001
28^{+1}-33^{+6} weeks(n=55)	23	41.8%	
34-36^{+6} weeks(n=33)	3	9.1%	

We observed that 100% neonates of Group A had respiratory distress syndrome. The percentage of neonates affected with RDS decreased as the gestational age increased(9.1% in the late preterm group) . This difference was statistically significant(p-value 0.001).

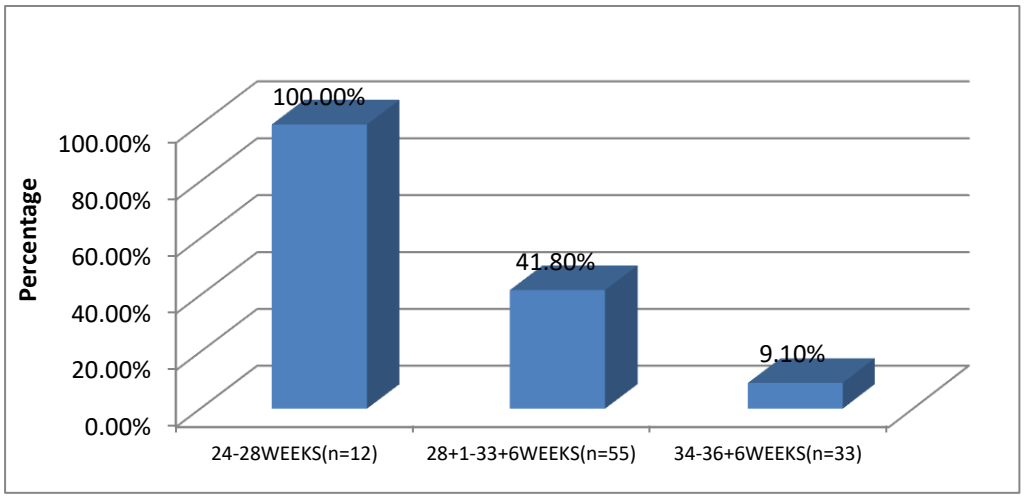


FIGURE 29: Comparison of respiratory distress syndrome in neonates of the three groups

TABLE 32: Comparison of TTNB in neonates of the three groups

Gestational age	Transient tachypnea of newborn (n=8)	Percentage	p-value
24-28 weeks(n=12)	0	0.0%	0.069
28^{+1}-33^{+6} weeks(n=55)	2	3.6%	
34-36^{+6} weeks(n=33)	6	18.2%	

In our study, we found that no newborn had TTNB at 24-28weeks of gestation. 2 out of 55 newborns (3.6%) of Group B had TTNB and 18.2% newborns of Group C had TTNB. This difference in the distribution of cases of TTNB among different gestational groups was not statistically significant (p-value 0.06).

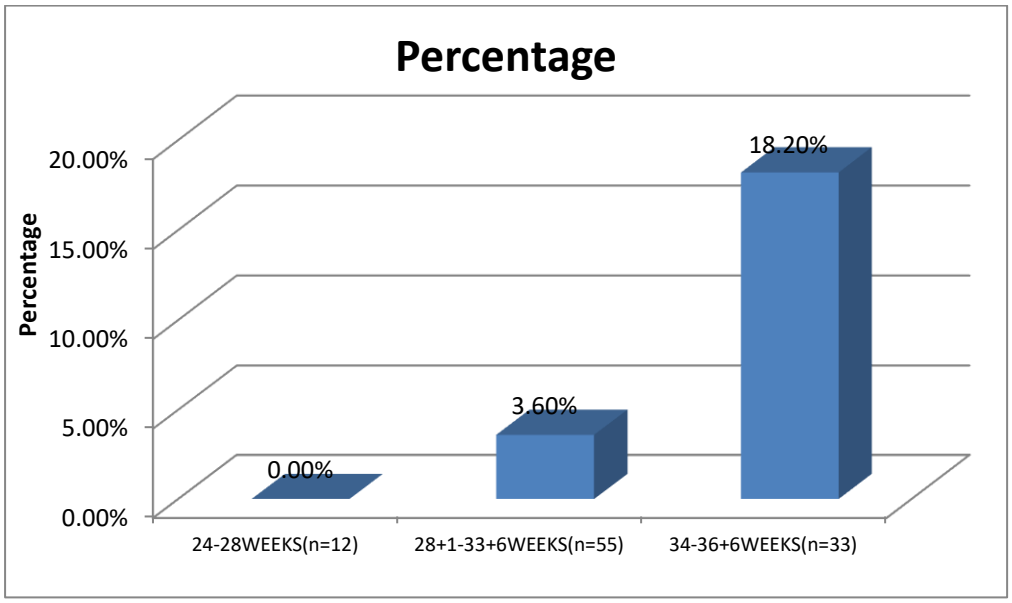


FIGURE 30: Comparison of TTNB in neonates of the three groups

TABLE 33: Comparison of congenital pneumonia in neonates of the three groups

Gestational age	Pneumonia(n=19)	Percentage	p-value
24-28 weeks(n=12)	0	0.0%	0.043
28^{+1}-33^{+6} weeks(n=55)	16	29.1%	
34-36^{+6} weeks(n=33)	3	9.1%	

It was seen that no newborn was reported to have congenital pneumonia at extreme preterm gestation in our study. From Group B, 29.1% of newborns had pneumonia and from Group C, three out of 33 newborns had pneumonia. The difference was statistically significant(p-value 0.04).

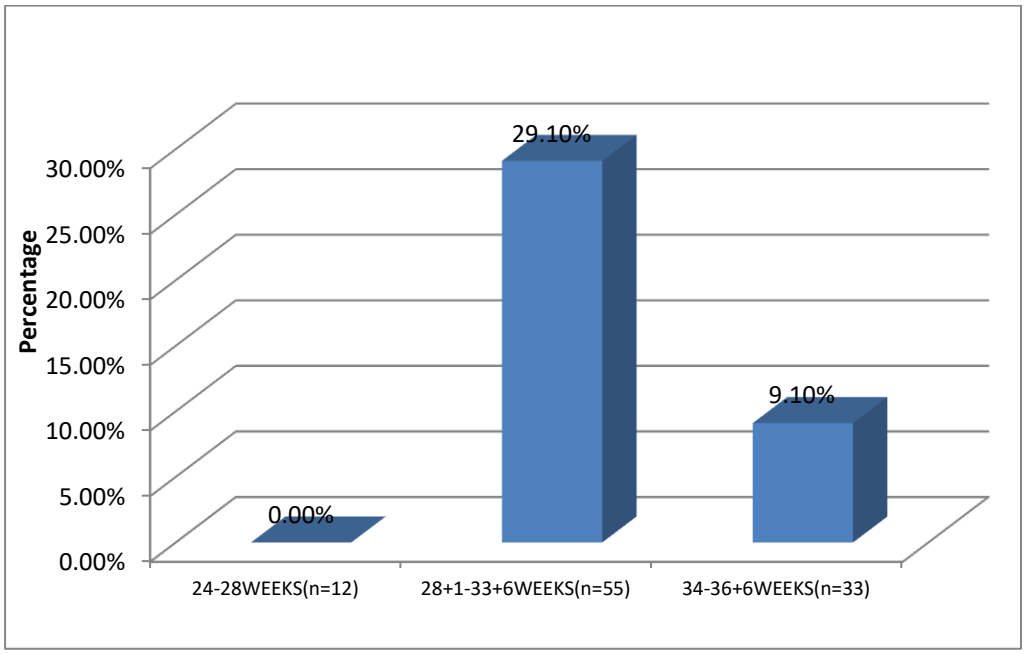


FIGURE 31: Comparison of congenital pneumonia in neonates of the three groups

DISCUSSION

Etiopathogenesis of preterm premature rupture of membranes and preterm birth is considered complex and heterogeneous, resulting from the interplay of several genetic and environmental factors. A very important risk factor for delivering a preterm neonate is a history of a prior preterm birth. The role of other predictors, such as maternal and/or genetic factors, environment or an interaction between these remains unknown. Fetoscopy may also carry a risk of iatrogenic PPROM.[45] PPROM is an important cause of perinatal morbidity and mortality. The most common complication among neonates is RDS. Other complications can be pneumothorax, bronchopulmonary dysplasia, pneumonia, necrotizing enterocolitis and intraventricular hemorrhage.

This study was conducted to determine the effect of preterm premature rupture of membranes on neonatal respiratory morbidity. A prospective clinical study was conducted in the department of Obstetrics and Gynaecology at Dayanand Medical College and Hospital for a time period of 2 years, from September 2019 to September 2021.

DEMOGRAPHIC PARAMETERS

In our study we observed that PPROM was present in a majority of women (40%) in the age group of 31-35 years and 7% women between 36-40 years of age. PPROM is evidenced more in advancing age. This can be explained as advanced maternal age is associated with adverse obstetric outcomes. In our study, the mean age of the women presenting with PPROM was 29years +- 4.43 (19-39years). Similar results were obtained by Berkowitz et al in their study about the risk factors for preterm birth in which they found that mothers 30 years of age or older had a significantly increased risk for PPROM.[46] A large population-based retrospective cohort study by Lucke & Brown documented the risks of maternal morbidity and adverse outcomes with increasing maternal age and a significantly higher risk of PPROM after adjusting for maternal race, parity, diabetes, chronic hypertension

and smoking status.[47] Similarly, Ziadeh et al in a large study conducted in Jordan found that women delivering their first child at age 35 years or older were at increased risk of PPROM compared with women aged 20-29 years of age. Ferguson et al also had a similar finding regarding maternal age.[48,49] It was seen that among patients less than 25 years (33.33%) and patients more than 36 years (16.67%) majority of cases of PPROM occurred at extreme preterm gestational age. PPROM was seen at relatively later gestational ages between the age group of 25-35 years. This difference in age at presentation of PPROM in late preterm group was statistically significant (p-value 0.001). To the best of our knowledge, no studies are available documenting the relationship between gestational age at the onset of PPROM and maternal age.

In our study, 52% women were graduates and 20% were post graduate. The rest 28% were either illiterate or had primary or secondary level education. Our institute is a tertiary care private hospital and majority of women attending the antenatal clinics are educated. This is in contrast to other studies. Noor et al in an observational study found that PPROM was more frequent among women with no or low education.[50] Low educational level is related to the probability of seeking antenatal care inappropriately. When compared among the patients having gestation less than 28 weeks at the presentation of PPROM, it was seen that percentage of patients increased as the education level decreased. The difference was found to be statistically significant (p-value- 0.04). No studies were available where the relationship between gestational age at onset of PPROM and education levels of women have been compared.

In our study, the majority of women (34%) were from a lower middle class family, 15% and 7% belonged to upper lower class and lower class respectively. Twenty percent women belonged to upper class and 24% belonged to upper middle class. Similar findings were documented by Polzin and Brady.[51] They asserted that the rates of

PPROM negatively correlated with the socioeconomic level. The factors that lead to PPROM in low socio-economic status include poor hygiene, malnutrition, anemia, stress, overexertion, high parity, recurrent genitourinary infections, etc. These factors lead to a decreased antibacterial activity in the amniotic fluid of patients. Economic factors like costs of transportation to the hospital and the cost of receiving medical care may restrict prenatal care in pregnant women with low socio-economic status.[52] It was seen that lower the socio-economic class, earlier was the gestation of presentation of PPROM. This difference was statistically significant (p-value 0.01). In contradiction to our study, Ortiz et al could not find any relation of statistical significance between women's socio-economic variables and the risk of having PPROM.[53]

In our study, 91% pregnancies were booked cases and only 9% were unbooked. This is explained by the fact that our institute being a tertiary care private hospital caters to booked patients and the overall percentage of unbooked patients remains very low throughout the year. These results are comparable to a study conducted by Shwetha Patil et al where the percentage of unbooked cases of PPROM was less, accounting for 31% and booked cases to 69%.[54] As per Government of India norms, every pregnant woman should have atleast 4 visits for antenatal checkup. Even 1 antenatal visit was associated with a 1.04% reduction in the probability of neonatal mortality and 1.07% reduction in probability of infant mortality.[55]

In our study, 52% women were primigravida and 48% were multigravida. Begum & Chowdhury in their study observed the incidence of PPROM in multigravida to be about 54.0 percent and Moretti and Sibai observed 71 percent incidence in multigravida.[56,57] No direct association of gravidity and PPROM was found in any study.

MATERNAL PARAMETERS

In our study, the most commonly encountered risk factor in women with PPROM was genitourinary infection which was present in 54 % patients. The second most common factor was a history of prior preterm birth (30%). These risk factors are directly associated with PPROM. During infections, there is a release of inflammatory mediators like IL-1, IL-6, and Tumor necrosis factor. These lead to an increase in metalloproteinase activity, which is responsible for collagen degradation. This results in a decrease in the tensile power of membranes and their rupture. Similar findings were stated by Kacerovsky et al. in their study that is,microorganisms in the amniotic fluid are present in approximately 30% of patients, based on the results of culture techniques , and in 50% of patients using a combination of culture and molecular microbiologic techniques.[58] The frequency of infection increases over time (latency period), such that when a patient with preterm PROM eventually goes into labor, microorganisms are detected in 75% of cases.[58] Recent reports have indicated that PPROM may be associated with sterile inflammation in the fetal membranes (an inflammatory condition mimicking infection with no evidence of microbial presence either through culture or molecular methods). It is hypothesized that certain risk factors of PPROM (ie smoking, bleeding), can produce sterile inflammation in the fetal membranes leading to an immunocompromised state and allowing for microbial invasion.[59] A history of preterm PROM is a major risk factor for preterm PROM or preterm labor in a subsequent pregnancy as documented by Mercer et al.[60] and Doody et al.[61] in their studies. In a large prospective study, the National Institute of Child Health & Human Development Maternal-Fetal Medicine Units Network found prior preterm birth and preterm birth due to preterm PROM to be associated with subsequent preterm birth due to preterm PROM. Those with a prior preterm birth at 23–27 weeks had a 27.1% risk of preterm birth subsequently (*P* value .001). Those with a history of preterm PROM had a 13.5% risk of preterm birth due to preterm PROM in a subsequent gestation (versus 4.1%, relative risk 3.3, *P*value.01) and a

13.5-fold higher risk of preterm PROM at less than 28 weeks in the subsequent gestation (1.8% versus 0.13%, *P* value .01). In our study, other risk factors associated with PPROM were short inter pregnancy interval, lifestyle habits like smoking, depression, anxiety and increased physical activity. In a separate publication ,the investigators found medical complications, work in pregnancy, symptomatic contractions, bacterial vaginosis, and low body mass index to be associated with preterm birth due to preterm PROM in nulliparas. Preterm PROM in multiparas was associated with prior preterm PROM, prior preterm birth due to preterm labor, and low body mass index. The presence of a short cervix (less than 25 mm by transvaginal ultrasound) was associated with preterm PROM in both nulliparas and multiparas.[13] A positive association has also been found between periodontitis and preterm birth in a meta-analysis of 17 observational studies on periodontal disease and preterm birth conducted in 2007 ($p<0.001$).[62]

In our study, 58% patients had a duration of leaking of less than 24 hours at admission while 42% had a history of leaking of more than 24hours.This directly reflects the patients' awareness about reporting to the hospital in case of an episode of leaking per vaginum. The median duration of leaking per vaginum at the time of admission was 22hours with a range between 1 hour and 72 hours. In a similar study carried out in the department of Obstetrics and Gynecology at Lady Hardinge Medical College and Smt Sucheta Kriplani Hospital, New Delhi,[44] it was seen that the median duration of leaking before admission to the hospital was 22 hours with the range between 8 and 38 hours.

It was seen that 55 patients had associated labour pains along with leaking in our study. The duration of labour pains was less than 24 hours in 45.5% patients and it was more than 24 hours in 54.5% patients. In PPROM, most of the patients go into labour within a few hours. There is a release of fetal fibronectin by amnion epithelial cells. The fibronectin then binds to a specific toll-like receptor in the amnion

mesenchymal cells and activates signaling cascades. These result in increased prostaglandin E synthesis (PGE2). Increased prostaglandin levels promote cervical ripening and uterine contractions. According to a similar study by Biswas PR et al, labour generally occurs within 24 hours of PPROM in 35-50 % , within 72 hours in 70 %, and 90% of patients deliver within two weeks.[63] American college of obstetrics and gynecology states that latency periods appear to be prolonged with second-trimester PPROM as compared with PROM during later gestational ages. However, 40–50% of patients with periviable PROM give birth within the first week and approximately 70–80% give birth within 2-5 weeks after membrane rupture.[1]

In our study, induction of labour was done in 45% of the patients while 55% patients went into spontaneous labour. The indications for induction of labor were oligohydramnios, clinical chorioamnionitis and abruptio placentae. A recent large randomized trial of 1,839 women that evaluated immediate delivery versus expectant management in patients with PPROM between 34 weeks of gestation and 36weeks 6 days of gestation suggested benefits to expectant management. There was no significant difference in the primary outcome that is, neonatal sepsis or in the secondary outcome of composite neonatal morbidity. Infants in the immediate delivery group had higher rates of respiratory distress (relative risk [RR], 1.6; 95% CI, 1.1–2.3) and mechanical ventilation (RR, 1.4; 95% CI, 1.0–1.8) and spent more days in intensive care (4 days versus 2 days). However, maternal adverse outcomes, such as hemorrhage and infection, were approximately two times higher with expectant management, although the rate of cesarean birth was lower.[64] In a similar study, Naef et al. compared induction of labour versus expectant management in a group of 120 patients with PPROM occurring at 34 until 37 weeks gestation and found a non-significant decrease of chorioamnionitis in the induction group without a significant difference in neonatal morbidity (including RDS) between both the groups.[65]

In our study, 47% patients presenting with PPROM delivered vaginally , 3% underwent instrumental delivery (forceps) and the rest 50% underwent a cesarean section. It was seen that 61.7% of the patients who delivered vaginally belonged to the early preterm group and this difference in the mode of delivery was statistically significant (p-value 0.04). Cesarean section was done in 50% patients for indications like fetal distress, abruptio placenta, cord prolapse, scar tenderness and breech presentation. This is consistent with the results of Gao *et al*. that previous CS delivery, fetal distress, and malpresentation account for more than 50% of all CS deliveries.[66] Similarly a cross sectional study of delivery mode involving 39 hospitals in 14 provinces of China in 2011 reported the rate of CS delivery in patients of PPROM to be 54.6%.[67] In a similar study by Rana M et al comparing mode of delivery with gestational age, it was seen that 100% of women delivered vaginally before 34 weeks of gestation. Out of the patients who were induced, majority underwent cesarean section after 34weeks. The rate of spontaneous labor increased as the gestational age at admission increased, the difference between the rate of spontaneous labor of 67% at 28 to 32 weeks and 86% at 34 to 36 weeks was statistically significant (p = 0.001).[44]

On analyzing the results of urine routine and urine culture taken prior to initiation of antibiotics, it was seen that 26% patients had an insignificant number of pus cells, 52% had more than 5 pus cells and the rest 22% had other components. 40% patients had no growth on urine culture, 54% had significant growth and 6% samples were contaminated. Out of the 54 samples with significant growth, it was seen that E.coli was present predominantly in 40.7% samples followed by Klebsiella (29.6%). UTI during pregnancy is significantly associated with the development of PPROM. This finding was consistent with the study performed by Singh et al.[68] The odds of PPROM were 2.62 times higher among those pregnant women with a history of UTI in pregnancy than those who did not have UTI (AOR = 2.62, 95% CI = 1.32–5.19).

The present study also indicated that a significant association is present between vaginal infection and PPROM. Significant growth was present in the HVS of 60 patients while 38 patients had no growth. On culture and sensitivity testing, it was seen that 50% patients had E.coli infection while only 3.3% had reports suggesting Staphylococcus aureus and mixed growth. Inflammatory cells produced by genital infections are involved in weakening of the fetal membranes among pregnant women thus causing PPROM. In a study by Assefa et al[69] the odds of PPROM was 5.30 times higher among pregnant women with abnormal vaginal discharge as compared to those who did not have abnormal vaginal discharge. This finding was in agreement with the study findings in Mekelle city, Ethiopia. However, a similar study[44] showed that the culture was positive in only 16% women. The most common organism grown was *Escherichia coli* (46.6%), followed by *Klebsiella*(33.3%), pseudomonas (13.3%) and coagulase negative *staphylococcus aureus*(6.6%). These culture positive patients were treated with antibiotics according to antibiotic sensitivity test results.

In our study, it was seen on per speculum examination that in 80% patients, the liquor was clear, watery in consistency and non foul smelling. In 12% patients, it was stained with meconium. The colour of amniotic fluid is clinically significant for deciding fetal prognosis as evidenced by a study conducted by Mazor et al.[70] Patients were classified according to the color of amniotic fluid into two groups: MSAF(meconium stained amniotic fluid) and clear amniotic fluid. A statistically significant higher rate of perinatal complications was found between 28 and 31 weeks gestation, and even a higher rate was noted between 32 and 36 weeks gestation in the MSAF group in comparison with patients with clear amniotic fluid [51% vs. 27.2%, respectively, P=0.003; 20% vs. 9.8%, respectively, P=0.0004]. In our study, the rest 8% patients had blood stained liquor which is most likely secondary to placental abruption in patients of PPROM. According to ACOG,[1] Vaginal bleeding or blood stained liquor should raise concern for abruptio placentae, which should prompt consideration of delivery, with

the decision based on fetal status, the amount of bleeding, and gestational age.

In our study, majority of the patients (42%) had mild oligohydramnios (AFI between 5-8cm), 25% patients had severe oligohydramnios (AFI less than 5cm) and 33% patients had normal AFI (8-18cm). Severe oligohydramnios was a recurrent indication for cesarean section. Ashraf et al. conducted a study on the association of low AFI with PPROM.[71] The women were divided into two groups according to amniotic fluid index of ≥5 cm and <5 cm. These women were followed and monitored up to delivery. There was no significant difference between the two groups, regarding neonatal morbidities, except for neonatal sepsis and neonatal death, which were higher in the amniotic fluid index <5 cm group (*p*-0.039). In the subgroups of two categories of gestational ages of 26–29weeks and 30–34+6 weeks, neonatal morbidities were higher in the amniotic fluid index <5 cm group. The results suggest that amniotic fluid index <5 cm should be considered as a warning sign for predicting poor prognosis of pregnancy complicated by preterm premature rupture of membranes.In a study by Piazze et al, the validity of AFI in the cases of PPROM has been evaluated. They suggested that AFI <5 cm at the time of patient admission can be a valuable prognostic factor in the management of the patients.[72] A retrospective study of pregnancies complicated with PPROM and its association with AFI was performed by Weissmann-Brenner A et al.[73] One hundred and two singleton pregnancies with PPROM formed the study population. The mean AFI in the PPROM and the control groups was 5.8+/-3.6 cm (0–18.5 cm) and 13.7+/-3.2 cm (7.3–24.4 cm), respectively (P-0.001). An AFI of 10 cm had sensitivity, specificity, positive and negative predictive values of 89.2%, 88.5%, 72.2% and 96%, respectively, in supporting the diagnosis of PPROM.Borna et al. reported that AFI <5 cm in patients of PPROM between 26 and 34 weeks of pregnancy, increases the risk of maternal infection and cesarean delivery, which is in accordance with the present study.[74]

Out of the 100 patients in our study, 54% had complete steroid cover before delivery. Twenty one patients could not receive the complete course as they delivered before the last dose could be given. After 34 weeks, antenatal corticosteroids were not given (n=25) following standard protocols. No rescue dose of steroids was given. The use of antenatal corticosteroids after preterm PROM has been evaluated in several clinical trials and has been shown to reduce neonatal mortality, respiratory distress syndrome, intraventricular hemorrhage, and necrotizing enterocolitis. According to ACOG,antenatal corticosteroids are not associated with increased risks of maternal or neonatal infection regardless of gestational age. A single course of corticosteroids is recommended for pregnant women between 24 weeks and 33weeks 6 days period of gestation. It may be considered for pregnant women who are at risk of preterm birth within 7 days, or present with PPROM, as early as 23 weeks of gestation.A retrospective cohort study and a secondary analysis of a prospective cohort study suggest that corticosteroids do not increase the risk of chorioamnionitis.[1] In the National Institute of Child Health & Human Development Maternal-Fetal Medicine Units Network trial, the incidence of RDS after conservatively managed preterm PROM was 41% when corticosteroids were not administered, despite adjunctive antibiotic administration.[13] Contrary to this finding, in a study done by A. Gezer et al,[75] corticosteroid prophylaxis was performed in 45% (*n* = 82) of 184 women below 34 gestational weeks. There was no difference in RDS rates between women who did and did not receive corticosteroid prophylaxis (chi square test, *p value* < 0.243)

FETAL PARAMETERS

In our study, out of 100 babies born, 42% were low birth weight , 29% were very low birth weight , 11% were extremely low birth weight and 18 babies had a weight of >2500grams. Low birth weight can be attributed to delivery at an early gestation due to PPROM. This is comparable to the study on PPROM conducted by Khan S et al[76] where, in 19 (31.67%) cases weight of newly born babies was found to

be only 1000-1499 gm. In another 37 (61.67%) cases weight at birth was 1500-2499 gm. It was only in 4 (6.67%) cases that weight at the time of birth was recorded to be >2500gm. Mean weight at birth was 1730.0±516.5gm and Median weight was (1000 - 3000) gm.

In our study, Apgar score was measured at 1 and 5 minutes of birth for all newborn babies. 66% newborns had a score of <7 at 1minute and 56% had a score of <7 at 5mins. It was seen that all extremely preterm babies had an Apgar of <7 at 1 & 5minutes while only 3 babies in the late preterm gestation had an Apgar of <7 at 5min. This difference was stastically significant at 1 and 5 minutes (p value 0.001). This is comparable to the study by A. Gezer et al[(75)] in which the relationship between the gestational age and Apgar scores in the 1st and 5th min was investigated and in less than or equal to 28 weeks, low Apgar scores (less than 5) in the 1st and 5th minute were seen in 78.8% and 41.1% of the patients respectively; while in more than or equal to 33 weeks, these rates were 19.1% and 4.5%, respectively(*p value* < 0.0001 and *p value* <0.0001, respectively). In this study, low (<5) 1 and 5 minute Apgar scores were found to be associated with NICU and positive pressure ventilation requirement, sepsis and respiratory distress syndrome. Similarly, in multiple regression analysis, 1 and 5 min Apgar scores and birth weight predicted NICU requirement and low 5 min Apgar scores predicted RDS development.In another study by Cnattingius S.et al, lower Apgar scores were associated with higher relative risks of neonatal death and greater absolute rate differences in neonatal mortality in all gestational age strata. Among infants born at 28 to 31 weeks, the adjusted absolute rate differences according to the 5-minute Apgar score, with those who had a score of 9 or 10 serving as the reference group, were 51.7 for a score of 0 or 1, 25.5 for a score of 2 or 3, 7.1 for a score of 4 to 6 and 1.2 for a score of 7 or 8. An increase in the Apgar score between 5 minutes and 10 minutes was associated with lower neonatal mortality than a stable Apgar score.[(77)]
We found that Apgar scores provided substantial information about the risk of neonatal morbidity among preterm infants across gestational-

age strata. Several studies challenge the view that the Apgar score may be of limited use in preterm infants. As Apgar score reflects the condition of the infant only shortly after birth, it may reflect a variety of underlying factors and preceding events. Apgar scoring is more challenging in preterm infants who, owing to immaturity, may appear less vigorous than term infants.[78] In agreement with other studies, we also found that the percentage of infants with low Apgar scores increased with decreasing gestational age.[79] Several components of the Apgar score - including respiratory effort, irritability, muscle tone, and color - may be influenced by gestational age.[78,79] Consequently, it has been argued that a low Apgar score in preterm infants may reflect biologic immaturity rather than fetal depression in an otherwise healthy infant.[78] However, Apgar scores may provide useful prognostic information for the survival of preterm infants.

In our study, out of 100 newborns, 62 were shifted to Neonatal ICU and the rest 38 neonates were either shifted to mother side or kept in observation room for a few hours and then shifted to mother side. All neonates (100%) born at <28weeks were shifted to Neonatal ICU while only 5 neonates (15%) in the period of gestation of 34-36weeks6days were shifted to NICU. From the early preterm group, NICU admissions were 81.8%. This difference in NICU admissions was statistically significant (p value -0.001). This may be due to low Apgar scores of the preterm babies at lower gestations. Similarly, in a study on PPROM by A Gezer et al, NICU requirement and duration of stay in the NICU was inversely correlated with gestational age. Mean duration of stay in the NICU was 15.8 +/- 20.6 and 2.3 +/- 1.1 days in less than equal to 28 gestational weeks and more than equal to 33 gestational weeks, respectively. The differences among the groups were statistically significant.[75]

In our study, we found the association of Respiratory distress syndrome, Transient tachypnea of newborn and Congenital Pneumonia with PPROM. RDS and TTNB are common causes leading to early

dyspnea in preterm infants delivered after PPROM. Similar findings were documented by Liu S et al. The mean gestational age in the study was (31.9 ± 2.2) weeks and birth weight was (1661 ± 501) g . Sixty-nine infants were diagnosed with TTN and 30 were diagnosed with RDS. There were significant differences in gestational age (29.5 ± 2.5) vs. (32.0 ± 3.2) weeks (P = 0.002) and birth weight (P = 0.001). Nine cases in the RDS group had Apgar score ≤ 7 while four cases in the TTN group had Apgar score ≤ 7 (P = 0.001). Preterm infants with RDS were characterized by younger gestational age, lower birth weight, severe acidosis and more complications. Preterm infants with TTN showed lower hypoxemia and oxygenation index.[80]

In our study, it was seen that respiratory distress syndrome was the most common respiratory complication. Neonatal respiratory distress syndrome (RDS) occurs due to the deficiency of surfactant. This can caused by either inadequate surfactant production or surfactant inactivation in immature lungs. Prematurity due to preterm premature rupture of membranes affects both these factors and thus, directly contributes to RDS. According to a study by Zanardo et al, RDS developed in 55.4% of the examined newborns from pregnancies complicated by PPROM,[81] whereas Joon Ho Lee et al. reported that RDS was diagnosed in 47% of the cases of PPROM.[82] In our study, the percentage of neonates affected with RDS decreased as the gestational age increased (9.1% in the late preterm group) . This difference was statistically significant (p-value 0.001). Similar findings were observed by R H Usher et al that the incidence of RDS rose with increasing degrees of prematurity. Among infants delivered vaginally, there were only 3 cases of RDS among 6,923 infants delivered after 38 weeks, while with more premature delivery the incidence rose from 0.8 per cent at 37 to 38 weeks to 64.3 per cent at 29 to 30 weeks of gestation.[83]

Hallak M et al conducted a study to find out the relationship between PPROM and RDS. Retrospective analysis yielded a group of 1395 patients who were delivered between 24 and 35 weeks gestation and

the frequencies of premature rupture of the membranes and respiratory distress syndrome by each gestational week were analyzed with a log linear multiway contingency table analysis. Of the 797 patients with premature rupture of the membranes 69% had RDS. Of the 287 patients without premature rupture of the membranes, 60.3% had RDS. RDS was more frequent with premature rupture of the membranes (p-value< 0.01).[84] Niesłuchowska-Hoxha A et al conducted a study aimed to investigate the cause of respiratory distress syndrome (RDS) in neonates from singleton pregnancies with PPROM between 24+0 and 36+6 weeks by using regression analysis for various factors. In 175 singleton pregnancies with PPROM, 95 cases of RDS (54%) were diagnosed. The results of logistic regression analysis showed that the following variables are closely correlated with RDS: female gender, antenatal steroids use, abnormal UA and MCA pulsatility index, fetal distress, maternal Hemoglobin.[85] However, we did not study the different causes of RDS in PPROM as mentioned in this study. A study conducted by Emma J Sims et al[86] demonstrated that after the confounding variables that were considered to be risk factors for RDS were controlled for, a multiple logistic regression analysis confirmed that PPROM was associated independently with a reduction in the frequency of RDS. According to their study, preterm rupture of the membranes served as a stressful stimulus to accelerate fetal glucocorticoid production. This in turn induced surfactant production in fetal lungs. This theory was supported by Bauer et al in their observation of infants who were delivered after prolonged rupture of the membranes.

In our study, transient tachypnea of newborn was seen in 8% of neonates. The difference of occurrence of TTNB at different gestational groups was not significant (p-value-0.06). TTNB is caused by a delay in the clearance of fetal lung fluid after birth, which leads to ineffective gas exchange, respiratory distress, and tachypnea .It is mostly seen in late preterm infants who are delivered by cesarean section, if it is done before the onset of labour. Pérez Molina JJ et al[87] conducted a case-

control study of 110 neonates with transient tachypnea and 110 newborns controls, between May and October 2000, in the Hospital Civil de Guadalajara Dr. Juan I. Menchaca. The frequency of transient tachypnea was 2 percent newborns alive. The risk factors that were associated with transient tachypnea were: low Apgar score, preterm premature rupture of amniotic membranes, cesarean section and masculine gender. In a study by Shimokaze T et al[88] however, no effect of PPROM was found on neonatal respiratory morbidity attributable to TTNB at 32–41 weeks of gestation. Additionally, no effect was found on respiratory morbidity with the time elapsed since PPROM.

Congenital pneumonia occurred in 19% neonates in our study. There was a significant difference found with respect to gestational age at PPROM and development of pneumonia (p-value 0.04). In addition to problems secondary to prematurity, neonatal infection has an important role in morbidity and mortality cases with PPROM. Gezer et al documented in their study that as gestational age increases, problems due to prematurity decrease and problems due to infection increase which was also seen in our study.[75] Preterm rupture of membranes predisposes the neonate to infections as maternal genital tract infection can lead to vertical transmission to the newborn and aspiration of bacteria in the amniotic fluid can cause pneumonia . Premature infants have a documented immune dysfunction putting them at a continued risk for development of sepsis and pneumonia. GR Evaldson et al studied the association of preterm premature rupture of the membranes (PPROM) and ascending infection in women. Bacteriological, histological and immunofluorescence methods were used to study infection at various levels in the birth canal.[89] The anaerobe Bacteroides fragilis, which is very infrequently isolated in normal pregnant women, was found in 33% women. Congenital pneumonia caused by group B streptococci and Haemophilus influenza was seen in 13% of neonates which is comparable to our study. Incoherence to our study, Shimokaze T et al asserted[88] that no

association was found between neonatal infection and pneumonia with PPROM, interval from membrane rupture to delivery, induction management or gestational age.

In our study, 35% neonates had no respiratory complications. It is most likely a result of the use of antenatal antibiotics, corticosteroids, early intervention and improved neonatal care. Three extremely pretern neonates however, could not survive despite adequate and timely management. The mechanism underlying these mortalities in this group can be explained by a combination of altered antenatal lung development, infection and the fetal inflammatory response syndrome (FIRS) induced by PPROM and associated chorioamnionitis.

SUMMARY AND CONCLUSION

This prospective study was conducted on 100 pregnant patients between 24 weeks to 36 weeks 6 days period of gestation presenting with preterm premature rupture of membranes in the department of obstetrics and gynaecology at Dayanand Medical College and Hospital, Ludhiana to determine the effect of PPROM on neonatal respiratory morbidity.

- The mean age of the patients presenting with PPROM was 29 years with range between 19 and 39 years. It was seen that among patients less than 25 years (33.33%) and patients more than 36 years (16.67%) majority of cases of PPROM occurred at extreme preterm gestational age. However no overall significant difference was found.
- The mean gestational age at presentation was 32.2+/- 3weeks with majority of patients (55%) between 28weeks1day and 33weeks 6days period of gestation.
- The majority of patients were educated and booked cases. It was seen that as the education level of patients decreased, the gestational age at presentation of PPROM also decreased and this difference was found to be statistically significant.
- Majority of patients (34%) belonged to a lower middle class family. Most of the patients who presented with leaking at less than 28weeks gestation belonged to upper lower and lower class. The difference in socio economic class and gestational age was found to be stastically significant.
- Primigravidity was seen in majority of patients (52%) but it had no direct association with PPROM.
- The most commonly encountered risk factor in patients with PPROM was genitourinary infection (32%). The second most common risk factor was a history of prior preterm birth (30%).
- The mean duration of leaking per vaginum at the time of admission to the hospital was 22hours with a range between 1 hour to 72

hours. This directly reflects the patients' awareness of leaking per vaginum and to report to the hospital in case of such an episode.

- Majority (55%) of patients had associated labour pains along with leaking per vaginum. In PPROM, about half of the patients went into labour within 24 hours of membrane rupture.
- Induction of labour was done in 45% of the patients. The indications for induction of labor were oligohydramnios, clinical chorioamnionitis and abruptio placentae.
- Half of the patients in our study (50%), underwent a cesarean section. Cesarean section was done for indications like fetal distress, massive abruption, cord prolapse, scar tenderness and breech presentation.
- On analyzing the results of urine routine and urine culture taken prior to initiation of antibiotics, it was seen that significant bacterial growth was present in 54% patients with E.coli being the most common organism involved. On High vaginal swab, 30% patients had infection due to E.coli. It indicates that a significant association is present between genitor-urinary infection and PPROM.
- In our study, 80% patients had clear liquor at presentation. Majority of our patients presented timely to the hospital befor onset of sepsis. This can be attributed to awareness of complications as majority were educated.
- Majority of the patients (42%) had mild oligohydramnios(AFI between 5-8cm) and 25% patients had severe oligohydramnios(AFI less than 5cm). As patients presented timely to the hospital so they were mostly with mild oligohydramnios.
- In our study, 54% patients had complete antenatal steroid cover before delivery. Steroids reduce neonatal mortality, respiratory distress syndrome, intraventricular hemorrhage, and necrotizing enterocolitis. Antenatal corticosteroids are a standard treatment protocol in patients with PPROM and DMC&H being a territory care centre, these standard protocols were followed.

- Majority of babies in our study were low birth weight (88%). Low birth weight can be attributed to delivery at an early gestation due to PPROM.
- It was seen that 100% of extremely preterm babies had an APGAR of <7 at 1 & 5 minutes while only 24% and 9% babies in the late preterm gestation had an APGAR of <7 at 1 and 5 min respectively. Problems due to prematurity along with high risk of fetal infection seem to responsible for this low Apgar score. This difference in Apgar score at 1 and 5 minutes at different periods of gestation was statistically significant .
- All neonates (100%) born at <28weeks were shifted to Neonatal ICU while only 5 neonates (15%) in the period of gestation of 34-36weeks6days were shifted to NICU. This difference in NICU admissions was statistically significant. This may be due to low Apgar scores of the preterm babies at lower gestations.
- In our study, respiratory distress syndrome was the most common respiratory complication seen in neonates. The percentage of neonates affected with RDS decreased as the gestational age increased (9.1% in the late preterm group). This difference was statistically significant.
- Transient tachypnea of newborn was seen in 8% of neonates. The difference of occurrence of TTNB at different gestational groups was not significant.
- Congenital pneumonia occurred in 19% neonates in our study. There was a significant difference found with respect to gestational age at PPROM and development of pneumonia implying that as the gestational age increases, problems due to prematurity decrease and problems due to infection increase. Neonatal infection has an important role in the development of pneumonia.

The limitation of statistical analysis was contributed by the small sample size within each gestational group in our study.

Based on all the observations, analyses and inferences it has been observed that since the risk of neonatal respiratory complications changes dramatically with gestational age at membrane rupture and

delivery, a gestational age based approach to the management of preterm PROM is appropriate. Looking after a premature infant puts immense burden on the economic and health care resources of the country. Therefore risk scoring strategies involving the demographic variables along with previous history of preterm deliveries should be developed to identify the high risk cases and treat them prior to rupture of membranes.

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Sr. No	MRD	NAME	AGE	DOA	BOOKED / UNBOOKED	EDUCATION	SOCIO-ECONOMIC STATUS	PRESENTING COMPLAINTS			OBSTETRICS HISTORY		MULTI	PRIMI
								H/O AMENORRHEA	2. h/o LEAKING SINCE(HOURS)	3. ASSOCIATED LABOR PAINS SINCE	LMP	EDD		
1	580812	K	26	7/9/2021	BOOKED	PG	UPPER CLASS	27WEEKS	12	5HOURS	2/3/2021	7/12/2021	Y	-
2	1169289	M	31	7/9/2021	BOOKED	PG	UPPER MIDDLE	33WEEKS6DAYS	4	NO	13/1/21	20/10/21	-	Y
3	1165591	S	32	3/9/2021	BOOKED	G	UPPER LOWER	33WEEKS5DAYS	10	NO	10/1/2021	17/10/21	Y	-
4	501877	S	26	4/9/2021	BOOKED	PG	UPPER CLASS	34WEEKS4DAYS	2	NO	5/1/2021	12/10/2021	-	Y
5	1160336	N	27	26/8/2021	BOOKED	PG	UPPER CLASS	36WEEKS6DAYS	4	12HOURS	11/12/2020	17/9/21	-	Y
6	1155986	K	23	17/8/2021	BOOKED	G	LOWER MIDDLE	30WEEKS	24	24HOURS	19/1/21	26/20/21	Y	-
7	1154823	B	24	14/8/2021	BOOKED	O	UPPER LOWER	31WEEKS2DAYS	24	26HOURS	7/1/2021	14/10/21	Y	-
8	1118612	N	28	19/8/2021	BOOKED	G	UPPER MIDDLE	35WEEKS1DAYS	12	8HOURS	16/12/20	22/09/21	-	Y
9	752760	K	23	15/8/2021	BOOKED	G	UPPER LOWER	32WEEKS4DAYS	4	NO	30/12/20	6/10/2021	Y	-
10	1103044	M	24	13/8/2021	UNBOOKED	G	UPPER MIDDLE	26WEEKS3DAYS	8	NO	9/2/2021	16/11/21	-	Y
11	1002823	R	24	13/8/2021	BOOKED	G	LOWER MIDDLE	33WEEKS	8	8HOURS	25/12/20	1/10/2021	Y	-
12	1100987	Y	20	14/8/2021	BOOKED	O	LOWER MIDDLE	31WEEKS5DAYS	2	NO	4/1/2021	11/10/2021	Y	-
13	1106740	P	36	10/8/2021	UNBOOKED	O	UPPER LOWER	25WEEKS6DAYS	48	48HOURS	10/2/2021	17/11/21	Y	-
14	767438	A	25	8/8/2021	BOOKED	G	LOWER MIDDLE	32WEEKS5DAYS	6	NO	22/12/20	28/9/21	Y	-
15	1108709	P	29	1/8/2021	BOOKED	PG	UPPER CLASS	36WEEKS6DAYS	1	NO	16/11/20	23/8/21	-	Y
16	1108234	H	26	24/7/2021	BOOKED	G	UPPER LOWER	31WEEKS	10	12HOURS	19/12/20	25/9/21	Y	-
17	1108099	A	30	20/7/2021	BOOKED	PG	UPPER CLASS	36WEEKS	26	24HOURS	10/11/2020	17/8/21	-	Y
18	1098231	K	27	16/7/2021	UNBOOKED	PG	UPPER MIDDLE	29WEEEKS4DAYS	12	NO	21/12/20	27/9/21	Y	-
19	1070981	M	33	1/7/2021	BOOKED	PG	UPPER CLASS	33WEEKS4DAYS	10	16HOURS	8/11/2020	15/8/21	Y	-
20	1053710	P	28	30/6/2021	BOOKED	PG	UPPER MIDDLE	29WEEKS1DAY	48	NO	8/12/2020	14/9/21	-	Y
21	1004155	S	26	20/6/2021	BOOKED	PG	UPPER MIDDLE	35WEEKS5DAYS	2	NO	13/10/20	20/7/21	Y	-
22	1002109	S	29	11/6/2021	BOOKED	G	LOWER MIDDLE	31WEEKS1DAY	18	20HOURS	5/11/2020	12/8/2021	Y	-
23	1000218	H	30	2/6/2021	BOOKED	G	UPPER CLASS	30WEEKS	12	12HOURS	4/11/2020	11/8/2021	-	Y
24	1000008	P	24	14/5/2021	BOOKED	G	UPPER MIDDLE	34WEEKS3DAYS	6	NO	15/9/20	22/6/21	-	Y
25	989860	R	24	30/4/2021	BOOKED	G	UPPER LOWER	27WEEKS6DAYS	1	NO	17/10/20	24/7/21	Y	-
26	987609	S	29	5/4/2021	BOOKED	G	UPPER MIDDLE	31WEEKS5DAYS	22	48HOURS	26/8/20	2/6/2021	Y	-
27	967541	R	39	1/4/2021	BOOKED	G	LOWER MIDDLE	34WEEKS1DAY	48	NO	5/8/2020	12/5/2021	-	Y
28	943098	P	28	25/3/2021	BOOKED	G	UPPER CLASS	30WEEKS	28	NO	27/8/20	3/6/2021	Y	-
29	977792	P	31	27/10/2020	BOOKED	O	LOWER MIDDLE	33WEEKS3DAYS	12	4HOURS	7/3/2020	12/12/2020	Y	-
30	992333	S	19	27/11/2020	UNBOOKED	O	LOWER	24WEEKS5DAYS	10	NO	7/6/2020	14/3/21	Y	-
31	186939	P	27	22/2/2021	BOOKED	G	LOWER MIDDLE	35WEEKS6DAYS	26	26HOURS	16/6/20	23/3/21	-	Y
32	1070030	S	28	31/3/2021	BOOKED	G	LOWER MIDDLE	35WEEKS	10	NO	29/7/20	5/5/2021	-	Y
33	1074733	E	27	4/4/2021	BOOKED	O	UPPER LOWER	26WEEKS6DAYS	1	NO	28/9/20	5/7/2021	Y	-
34	1111742	M	31	22/5/2021	BOOKED	G	UPPER MIDDLE	32WEEKS	3	NO	10/10/2020	17/7/21	Y	-
35	1113310	P	29	26/5/2021	UNBOOKED	G	LOWER MIDDLE	36WEEKS3DAYS	4	10HOURS	13/9/20	20/6/21	-	Y
36	1110590	P	32	27/5/2021	BOOKED	G	UPPER MIDDLE	30WEEKS1DAY	18	NO	28/10/20	4/8/2021	-	Y
37	996182	P	23	16/12/2020	BOOKED	O	UPPER MIDDLE	34WEEKS5DAYS	48	12HOURS	17/4/20	22/1/21	-	Y
38	1005948	K	33	28/12/2020	BOOKED	O	LOWER MIDDLE	31WEEKS4DAYS	48	12HOURS	21/5/20	25/2/21	Y	-

Sr. No	MRD	H/O OF PPROM IN PREVIOUS PREGNANCIES	GENERAL PHYSICAL EXAMINATION				COLOUR	INVESTIGATION					
			BMI	B.P	PR	TEMPERATURE		Hb%	TLC	CRP	URINE-R	HIGH VAGINAL SWAB C/S	URINE C/S
1	580812	NO	22.6	110/70	88	98	CLEAR	12.2	7.8	2	NAD	NO GROWTH	NO GROWTH
2	1169289	-	23	100/70	106	99.6	MECONIUM	11.6	17	8.5	NAD	NO GROWTH	ENTEROBACTER CLOACAE 10^3
3	1165591	YES	20.8	100/70	96	98.6	BLOOD TINGED	9.9	15.2	20	NAD	E.COLI ++	NO GROWTH
4	501877	-	23	110/76	80	98	CLEAR	11	8.2	6.2	4-5PUS CELLS	NO GROWTH	NO GROWTH
5	1160336	-	22.8	100/60	98	98.4	CLEAR	12	9	3.1	NAD	NO GROWTH	NO GROWTH
6	1155986	YES	19.8	100/60	102	99	CLEAR	10.4	11	24	10-12PUS CELLS	NO GROWTH	E.COLI 10^5
7	1154823	YES	23	120/80	76	97.4	CLEAR	12.8	8.4	8	NAD	KLEBSIELLA PNEMONIA ++	NO GROWTH
8	1118612	-	21	110/70	80	98	CLEAR	11.2	9.8	0.9	NAD	NO GROWTH	NO GROWTH
9	752760	YES	24.4	120/80	98	97.6	CLEAR	13.5	10	15	6-7PUS CELLS	E.COLI ++	NO GROWTH
10	1103044	-	19	90/60	104	98.8	CLEAR	9.8	14.4	6	20-40PUS CELLS	NO GROWTH	E.COLI 10^5
11	1002823	YES	22.8	120/70	88	98.2	CLEAR	11.6	8.9	1.2	NAD	NO GROWTH	NO GROWTH
12	1100987	NO	25.3	120/80	70	99.2	CLEAR	13	9	5.2	CANDIDA +	NO GROWTH	NO GROWTH
13	1106740	YES	20	100/60	112	100.4	CLEAR	9	19.1	35	BACTERIA+	ENTEROCOCCUS FAECIUM +	PSEUDOMONAS AERUGINOSA 10^5
14	767438	NO	21	100/70	98	97.8	CLEAR	11.4	8.5	0.8	NAD	NO GROWTH	NO GROWTH
15	1108709	-	19.8	100/68	90	98.4	CLEAR	10	8.7	1.4	NAD	CANDIDA+	NO GROWTH
16	1108234	NO	23.6	120/80	72	97.4	CLEAR	12.9	10	1.6	NAD	NO GROWTH	NO GROWTH
17	1108099	-	22.7	110/78	88	100	MECONIUM	12	20.8	22	4-5PUS CELLS	E.COLI ++	E.COLI 10^5
18	1098231	YES	18.8	90/60	100	97	CLEAR	10.5	10.8	1.4	NAD	NO GROWTH	NO GROWTH
19	1070981	YES	21.8	120/78	80	98.4	CLEAR	12.8	7.4	2.2	NAD	NO GROWTH	NO GROWTH
20	1053710	-	23	110/76	90	98.2	CLEAR	12	20.7	25.3	NAD	NO GROWTH	PROTEUS SP. 10^5
21	1004155	NO	20.7	120/76	92	99	MECONIUM	11	12.8	9.7	YEAST+	NO GROWTH	NO GROWTH
22	1002109	NO	26.8	110/60	80	98.6	CLEAR	10.8	11	3.2	NAD	NO GROWTH	NO GROWTH
23	1000218	-	21	110/70	74	98.4	CLEAR	11.2	7.9	5.9	NAD	CANDIDA++	NO GROWTH
24	1000008	-	23.9	120/80	86	98	CLEAR	12.4	8	2.7	NAD	NO GROWTH	NO GROWTH
25	989860	YES	20.1	110/70	90	98.4	MECONIUM	10	17.1	10.8	BACTERIA+	NO GROWTH	PSEUDOMONAS AERUGINOSA 10^5
26	987609	YES	24.2	120/80	98	99.2	CLEAR	10.2	20	15.6	YEAST+	CANDIDA+	NO GROWTH
27	967541	-	22.6	120/78	100	98.6	CLEAR	11.4	16	4.5	NAD	NO GROWTH	NO GROWTH
28	943098	NO	18.6	120/70	86	98	CLEAR	12.1	11.5	2.1	NAD	NO GROWTH	NO GROWTH
29	977792	YES	26	110/72	88	98.2	CLEAR	10.8	14	8.9	NAD	E.COLI ++	NO GROWTH
30	992333	NO	28.2	120/80	90	98.6	CLEAR	12	14.8	3.6	7-10PUS CELLS	NO GROWTH	NO GROWTH
31	186939	-	25.8	110/78	78	98.6	BLOOD TINGED	10.6	9.1	2.2	NAD	NO GROWTH	NO GROWTH
32	1070030	-	22.2	110/70	102	99.4	MECONIUM	12	8.1	0.8	NAD	NO GROWTH	NO GROWTH
33	1074733	YES	20.6	120/78	68	97.6	CLEAR	11.1	6.9	3.3	NAD	KLEBSIELLA +	NO GROWTH
34	1111742	YES	18.3	120/72	92	98.4	CLEAR	10	12.9	6.7	BACTERIA+	NO GROWTH	E.COLI 10^5
35	1113310	-	23.6	120/80	88	98	CLEAR	13.2	15.1	3.5	NAD	NO GROWTH	NO GROWTH
36	1110590	-	21	110/70	114	100.2	CLEAR	10	21.4	16.7	YEAST+	ENTEROCOCCUS FAECIUM++	NO GROWTH
37	996182	-	19.7	100/60	78	97.4	BLOOD TINGED	9.8	12	2.2	NAD	NO GROWTH	NO GROWTH
38	1005948	YES	19	110/70	86	98.2	MECONIUM	11.2	11	5.4	NAD	NO GROWTH	NO GROWTH

Sr. No	MRD	USG (AFI)	ACTIVE MANAGEMENT			ANTIBIOTICS	STEROIDS	FETAL OUTCOME				SHIFTED TO NICU / ROOMING	ASSOCIATED RESPIRATORY MORBIDITY			FETAL MORTALITY
			MODE OF DELIVERY					SEX	BIRTH WEIGHT(GRAMS)	APGAR SCORE						
			NORMAL	INSTRUMENTAL	LSCS					1 Min	5 Min		RDS	TTNB	PNEUMONIA	
1	580812	12CM	Y	-	-	YES	INCOMPLETE	MALE	875	5	6	NICU	Y	-	-	-
2	1169289	3-4CM	-	-	Y	YES	COMPLETE	FEMALE	2100	8	9	ROOM	-	Y	-	-
3	1165591	1CM	-	-	Y	YES	COMPLETE	MALE	2050	8	9	ROOM	-	-	-	-
4	501877	10CM	Y	-	-	YES	NO	MALE	2030	8	9	NICU	Y	-	-	-
5	1160336	15CM	-	-	Y	YES	NO	FEMALE	2680	9	9	ROOM	-	-	-	-
6	1155986	8CM	Y	-	-	YES	INCOMPLETE	MALE	1330	5	6	NICU	Y	-	-	-
7	1154823	13CM	-	-	Y	YES	COMPLETE	MALE	1540	5	6	NICU	-	-	Y	-
8	1118612	7-8CM	-	-	Y	YES	NO	MALE	2320	8	9	ROOM	-	-	-	-
9	752760	18CM	-	Y	Y	YES	COMPLETE	FEMALE	1800	8	9	ROOM	-	-	-	-
10	1103044	6CM	Y	-	-	YES	INCOMPLETE	MALE	809	5	6	NICU	Y	-	-	-
11	1002823	10CM	-	-	Y	YES	COMPLETE	FEMALE	1900	6	7	NICU	-	-	Y	-
12	1100987	12CM	Y	-	-	YES	COMPLETE	MALE	1630	6	7	ROOM	-	-	-	-
13	1106740	1CM	Y	-	-	YES	INCOMPLETE	MALE	740	3	4	NICU	Y	-	-	YES
14	767438	9CM	-	-	Y	YES	COMPLETE	FEMALE	1870	7	7	NICU	-	-	Y	-
15	1108709	10CM	-	-	-	YES	NO	FEMALE	2780	8	9	ROOM	-	-	-	-
16	1108234	13CM	Y	-	-	YES	INCOMPLETE	FEMALE	1520	6	6	NICU	-	-	Y	-
17	1108099	0CM	-	-	Y	YES	NO	MALE	2530	8	9	ROOM	-	-	-	-
18	1098231	10CM	Y	-	-	YES	INCOMPLETE	MALE	1250	5	6	NICU	Y	-	-	-
19	1070981	20CM	-	-	Y	YES	COMPLETE	MALE	2080	8	9	ROOM	-	-	-	-
20	1053710	6CM	Y	-	-	YES	COMPLETE	FEMALE	1180	5	6	NICU	Y	-	-	-
21	1004155	12CM	Y	-	Y	YES	NO	MALE	2380	8	8	ROOM	-	Y	-	-
22	1002109	8CM	-	-	Y	YES	COMPLETE	MALE	1520	6	6	NICU	-	-	Y	-
23	1000218	19CM	Y	-	-	YES	INCOMPLETE	FEMALE	1320	5	6	NICU	Y	-	-	-
24	1000008	15CM	Y	-	-	YES	NO	FEMALE	2000	6	6	NICU	Y	-	-	-
25	989860	9CM	-	-	Y	YES	COMPLETE	FEMALE	1041	6	6	NICU	Y	-	-	-
26	987609	7CM	-	-	Y	YES	INCOMPLETE	MALE	1600	6	6	ROOM	-	-	-	-
27	967541	19CM	Y	-	-	YES	COMPLETE	FEMALE	1980	5	6	NICU	Y	-	-	-
28	943098	16CM	Y	-	-	YES	COMPLETE	FEMALE	1340	5	6	NICU	Y	-	-	-
29	977792	7CM	-	-	Y	YES	COMPLETE	MALE	2020	8	9	ROOM	-	-	-	-
30	992333	6CM	Y	-	-	YES	INCOMPLETE	FEMALE	650	3	4	NICU	Y	-	-	YES
31	186939	18CM	-	-	Y	YES	NO	FEMALE	2420	9	9	ROOM	-	-	-	-
32	1070030	15CM	-	-	Y	YES	COMPLETE	MALE	2400	9	9	ROOM	-	Y	-	-
33	1074733	12CM	Y	-	-	YES	INCOMPLETE	MALE	860	5	6	NICU	Y	-	-	-
34	1111742	14CM	-	-	Y	YES	COMPLETE	MALE	1650	6	7	NICU	-	-	Y	-
35	1113310	11CM	-	Y	-	YES	NO	MALE	2650	9	9	ROOM	-	-	-	-
36	1110590	5CM	Y	-	-	YES	COMPLETE	MALE	1560	5	6	NICU	-	-	Y	-
37	996182	9CM	-	-	Y	YES	INCOMPLETE	FEMALE	2140	8	9	ROOM	-	-	-	-
38	1005948	9CM	-	-	Y	YES	COMPLETE	MALE	1500	5	6	NICU	-	-	Y	-

Sr. No	MRD	NAME	AGE	DOA	BOOKED / UNBOOKED	EDUCATION	SOCIO-ECONOMIC STATUS	PRESENTING COMPLAINTS			OBSTETRICS HISTORY		MULTI	PRIMI
								H/O AMENORRHEA	2. h/o LEAKING SINCE(HOURS)	3. ASSOCIATED LABOR PAINS SINCE	LMP	EDD		
39	1033575	M	34	24/2/2021	BOOKED	PG	UPPER MIDDLE	31WEEKS1DAY	24	2HOURS	21/7/20	27/4/21	-	Y
40	1023869	H	30	7/2/2021	BOOKED	O	UPPER CLASS	35WEEKS1DAY	12	8HOURS	6/6/2020	13/3/21	Y	-
41	937226	T	23	29/1/2021	BOOKED	O	LOWER MIDDLE	34WEEKS6DAYS	6	NO	30/5/20	6/3/2021	-	Y
42	984344	R	34	7/11/2020	BOOKED	G	LOWER MIDDLE	33WEEKS5DAYS	36	36HOURS	16/3/20	21/12/20	Y	-
43	451978	K	29	2/11/2020	BOOKED	O	LOWER	27WEEKS	10	NO	27/4/20	1/2/2021	-	Y
44	1024686	R	20	8/2/2021	BOOKED	G	LOWER MIDDLE	30WEEKS4DAYS	30	5HOURS	9/7/2020	15/4/21	-	Y
45	898528	G	26	17/2/2020	BOOKED	O	LOWER MIDDLE	34WEEKS4DAYS	26	10HOURS	20/6/19	26/3/20	Y	-
46	568535	K	24	26/12/2020	BOOKED	G	LOWER MIDDLE	32WEEKS5DAYS	12	32HOURS	11/5/2020	15/2/21	Y	-
47	705782	S	27	29/12/2020	BOOKED	G	UPPER MIDDLE	36WEEKS6DAYS	11	10HOURS	15/4/20	20/1/21	-	Y
48	1004435	P	27	30/1/2021	BOOKED	G	LOWER MIDDLE	30WEEKS4DAYS	4	NO	30/6/20	6/4/2021	-	Y
49	997874	M	31	11/12/2020	UNBOOKED	G	UPPER MIDDLE	27WEEKS1DAY	48	NO	4/6/2020	11/3/2021	-	Y
50	783461	H	23	7/10/2020	BOOKED	G	LOWER MIDDLE	32WEEKS5DAYS	11	6HOURS	21/2/20	27/11/20	Y	-
51	955347	S	31	21/12/2020	BOOKED	O	LOWER MIDDLE	34WEEKS5DAYS	20	8HOURS	22/4/20	27/1/21	-	Y
52	1031356	M	35	24/2/2021	BOOKED	G	LOWER MIDDLE	30WEEKS1DAY	36	NO	28/7/20	4/5/2021	Y	-
53	1110016	N	32	21/5/2021	BOOKED	G	UPPER LOWER	35WEEKS5DAYS	36	6HOURS	13/9/20	20/6/21	-	Y
54	1109457	I	35	18/5/2021	BOOKED	O	LOWER	33WEEKS6DAYS	36	28HOURS	23/9/20	30/6/21	Y	-
55	893558	S	32	16/5/2021	BOOKED	G	LOWER MIDDLE	31WEEKS	5	NO	11/10/2020	18/7/21	Y	-
56	1015048	S	33	3/5/2021	BOOKED	G	UPPER MIDDLE	35WEEKS2DAYS	7	NO	29/8/20	5/6/2021	-	Y
57	1002004	D	38	18/3/2021	BOOKED	PG	UPPER MIDDLE	30WEEKS	12	NO	20/8/20	27/5/21	-	Y
58	870943	M	32	11/6/2020	BOOKED	G	UPPER LOWER	30WEEKS3DAYS	40	10HOURS	11/11/2019	17/8/20	Y	-
59	667246	J	34	1/5/2020	BOOKED	G	LOWER MIDDLE	35WEEKS4DAYS	48	6HOURS	26/8/19	1/6/2020	Y	-
60	667092	R	39	30/4/2020	UNBOOKED	O	LOWER	24WEEKS4DAYS	7	NO	10/11/2019	16/8/20	-	Y
61	610294	M	31	2/3/2020	BOOKED	O	LOWER MIDDLE	33WEEKS3DAYS	72	26HOURS	12/7/2019	17/4/20	-	Y
62	600323	J	32	11/2/2020	BOOKED	PG	UPPER MIDDLE	32WEEKS3DAYS	4	NO	29/6/19	4/4/2020	-	Y
63	600018	A	35	8/2/2020	BOOKED	O	UPPER CLASS	36WEEKS5DAYS	48	24HOURS	27/5/19	2/3/2020	Y	-
64	597987	P	26	1/2/2020	BOOKED	G	LOWER MIDDLE	30WEEKS1DAY	18	NO	5/7/2019	10/4/2020	-	Y
65	596107	B	26	7/2/2020	BOOKED	G	LOWER MIDDLE	32WEEKS	72	32HOUR	28/6/19	3/4/2020	-	Y
66	583451	S	27	30/1/2020	BOOKED	O	LOWER	29WEEKS5DAYS	72	36HOURS	6/7/2019	11/4/2020	Y	-
67	565679	D	32	23/1/2020	BOOKED	PG	UPPER CLASS	36WEEKS4DAYS	48	40HOURS	12/5/2019	16/2/20	-	Y
68	565214	T	28	23/1/2020	BOOKED	O	LOWER MIDDLE	30WEEKS6DAYS	12	32HOURS	21/6/19	27/3/20	Y	-
69	554919	S	29	9/1/2020	BOOKED	O	LOWER MIDDLE	31WEEKS3DAYS	48	40HOURS	3/6/2019	9/3/2020	-	Y
70	549980	G	39	8/1/2020	BOOKED	G	LOWER	29WEEKS	20	29HOURS	19/6/19	25/3/20	-	Y
71	543091	R	31	6/1/2020	UNBOOKED	O	UPPER MIDDLE	34WEEKS6DAYS	10	NO	7/5/2019	11/2/2020	Y	-
72	500289	S	32	31/12/2019	BOOKED	PG	UPPER MIDDLE	36WEEKS3DAYS	2	NO	20/4/19	25/1/20	-	Y
73	487999	K	30	31/12/2019	BOOKED	O	LOWER MIDDLE	32WEEKS4DAYS	40	40HOURS	17/5/19	21/2/20	-	Y
74	478941	G	33	28/12/2019	BOOKED	O	UPPER LOWER	34WEEKS2DAYS	4	NO	2/5/2019	6/2/2020	Y	-
75	467610	K	30	25/12/2019	BOOKED	G	UPPER LOWER	31WEEKS2DAYS	24	28HOUR	20/5/19	24/2/20	Y	-
76	1034403	A	32	22/9/2021	BOOKED	PG	UPPER CLASS	33WEEKS6DAYS	10	5HOURS	28/1/21	4/11/2021	Y	-

Sr. No	MRD	H/O OF PPROM IN PREVIOUS PREGNANCIES	GENERAL PHYSICAL EXAMINATION				COLOUR	INVESTIGATION					
			BMI	B.P	PR	TEMPERATURE		Hb%	TLC	CRP	URINE-R	HIGH VAGINAL SWAB C/S	URINE C/S
39	1033575	-	20.6	100/70	80	98	CLEAR	12.3	10.8	2.4	NAD	NO GROWTH	NO GROWTH
40	1023869	NO	18	100/60	76	97.4	CLEAR	9.4	15.2	8.7	20-22PUS CELLS	NO GROWTH	E.COLI 10^5
41	937226	-	19.7	100/60	80	98.6	MECONIUM	11	14.6	3.9	NAD	CANDIDA++	NO GROWTH
42	984344	YES	26	110/70	98	98	CLEAR	12.8	8.7	6.6	6-7PUS CELLS	NO GROWTH	E.COLI 10^3
43	451978	-	23.5	100/70	76	98.6	CLEAR	11	9.8	1.2	NAD	E.COLI ++	NO GROWTH
44	1024686	-	20	120/80	80	98.4	CLEAR	13.2	8.8	3.2	NAD	NO GROWTH	NO GROWTH
45	898528	YES	19.8	110/74	98	99	CLEAR	11.3	19	10.4	YEAST+	CANDIDA +++	NO GROWTH
46	568535	NO	27.2	120/78	76	98.6	CLEAR	10.5	12.8	0.5	NAD	NO GROWTH	NO GROWTH
47	705782	-	22	120/80	86	97.6	CLEAR	10.8	16.7	8.6	NAD	NO GROWTH	NO GROWTH
48	1004435	-	21.7	110/70	90	98.6	CLEAR	12.4	11.2	4.8	BACTERIA+	NO GROWTH	PROTEUS SP. 10^5
49	997874	-	17.2	90/60	112	99.8	MECONIUM	9.6	20	17.8	70-80PUS CELLS	NO GROWTH	PSEUDOMONAS AERUGINOSA 10^5
50	783461	YES	27.8	120/78	78	97.2	CLEAR	12.9	5.6	2	NAD	NO GROWTH	NO GROWTH
51	955347	-	23.7	110/76	94	98.4	CLEAR	11.7	8	1.6	NAD	NO GROWTH	NO GROWTH
52	1031356	NO	20	110/70	108	99.6	CLEAR	10	19.3	18.1	10-12PUS CELLS	KLEBSIELLA PNEMONIA ++	NO GROWTH
53	1110016	-	18.2	100/60	96	98.2	CLEAR	11.2	7.8	1.1	NAD	NO GROWTH	NO GROWTH
54	1109457	NO	28.4	110/78	70	100.4	MECONIUM	9.9	18.9	33	YEAST+ BACTERIA+	NO GROWTH	E.COLI 10^5
55	893558	YES	21.8	100/70	104	98.6	CLEAR	11	15.2	7.6	NAD	NO GROWTH	NO GROWTH
56	1015048	-	22	110/70	86	97.6	CLEAR	13	10.2	2.4	NAD	NO GROWTH	NO GROWTH
57	1002004	-	26	100/72	120	99	CLEAR	10.2	20.5	8.9	24-25PUS CELLS	NO GROWTH	NO GROWTH
58	870943	YES	19.7	110/70	78	98.4	CLEAR	12.2	9	0.7	NAD	NO GROWTH	NO GROWTH
59	667246	NO	19	100/60	98	98	CLEAR	10.7	8.8	0.9	NAD	NO GROWTH	NO GROWTH
60	667092	-	21.6	110/70	80	98.6	CLEAR	11.2	9.1	2.5	YEAST+	CANDIDA +++	NO GROWTH
61	610294	-	20.7	120/80	90	98.4	CLEAR	12.6	11.2	5.2	NAD	NO GROWTH	NO GROWTH
62	600323	-	21.1	110/72	86	97.6	CLEAR	10.1	13.7	4.5	7-8PUS CELLS	NO GROWTH	PROTEUS SP. 10^5
63	600018	YES	23.2	110/76	92	98.4	CLEAR	11.5	12	3.7	NAD	STAPHYLOCOCCUS AUREUS +	NO GROWTH
64	597987	-	19	100/60	98	98	CLEAR	10.3	19.5	6	BACTERIA+	NO GROWTH	PSEUDOMONAS AERUGINOSA 10^5
65	596107	-	21.9	110/70	82	99	CLEAR	12.1	10	5.6	NAD	NO GROWTH	NO GROWTH
66	583451	YES	20.2	110/70	78	97.6	CLEAR	11.3	15.6	3.6	20-25PUS CELLS	NO GROWTH	NO GROWTH
67	565679	-	20.7	110/70	88	98.2	BLOOD TINGED	12	20.1	5.6	NAD	KLEBSIELLA SPECIES ++	NO GROWTH
68	565214	NO	23.1	120/80	80	98.6	CLEAR	12.5	10.1	1.8	NAD	NO GROWTH	NO GROWTH
69	554919	-	19.5	110/70	122	100.1	CLEAR	11.8	28	25	BACTERIA+	NO GROWTH	E.COLI 10^5
70	549980	-	18.8	100/70	86	97.6	CLEAR	12.5	6.4	1.5	NAD	NO GROWTH	NO GROWTH
71	543091	YES	23	120/80	78	98.4	BLOOD TINGED	11.6	8.7	6.7	NAD	E.COLI ++	NO GROWTH
72	500289	-	19	110/70	86	98	CLEAR	9	12.9	1.6	NAD	NO GROWTH	NO GROWTH
73	487999	-	20.8	110/80	72	97	CLEAR	10.1	10.5	1.8	YEAST+	NO GROWTH	NO GROWTH
74	478941	YES	19.7	100/70	96	98.4	CLEAR	9.5	11.2	4.3	NAD	NO GROWTH	NO GROWTH
75	467610	NO	18.6	110/72	84	98.2	CLEAR	10.3	9.9	5.2	NAD	NO GROWTH	NO GROWTH
76	1034403	YES	22.2	120/70	96	98	CLEAR	11.6	12	3.1	NAD	NO GROWTH	NO GROWTH

Sr. No	MRD	USG (AFI)	ACTIVE MANAGEMENT			ANTIBIOTICS	STEROIDS	FETAL OUTCOME				SHIFTED TO NICU / ROOMING	ASSOCIATED RESPIRATORY MORBIDITY			FETAL MORTALITY
			MODE OF DELIVERY					SEX	BIRTH WEIGHT(GRAMS)	APGAR SCORE						
			NORMAL	INSTRUMENTAL	LSCS					1 Min	5 Min		RDS	TTNB	PNEUMONIA	
39	1033575	10CM	Y	-	-	YES	COMPLETE	FEMALE	1450	5	6	NICU	Y	-	-	-
40	1023869	10CM	Y	-	-	YES	INCOMPLETE	FEMALE	2260	8	9	ROOM	-	-	-	-
41	937226	4CM	-	-	Y	YES	COMPLETE	MALE	2100	7	8	NICU	-	-	-	-
42	984344	16CM	-	-	Y	YES	COMPLETE	FEMALE	2010	8	9	ROOM	-	-	-	-
43	451978	6CM	Y	-	-	YES	COMPLETE	MALE	900	4	6	NICU	Y	-	-	-
44	1024686	12CM	Y	-	-	YES	INCOMPLETE	FEMALE	1370	5	6	NICU	Y	-	-	-
45	898528	10CM	-	-	Y	YES	COMPLETE	FEMALE	2050	6	6	NICU	-	-	-	-
46	568535	17CM	-	-	Y	YES	COMPLETE	MALE	1840	7	8	ROOM	-	-	-	-
47	705782	12CM	-	Y	-	YES	NO	MALE	2830	9	9	ROOM	-	-	-	-
48	1004435	3CM	Y	-	-	YES	INCOMPLETE	MALE	1840	7	8	ROOM	-	-	-	-
49	997874	NIL	-	-	Y	YES	COMPLETE	MALE	910	5	6	NICU	Y	-	-	-
50	783461	18CM	-	-	Y	YES	COMPLETE	FEMALE	1780	7	7	ROOM	-	-	-	-
51	955347	10CM	-	-	Y	YES	COMPLETE	MALE	2100	7	8	NICU	-	-	-	-
52	1031356	3CM	Y	-	-	YES	INCOMPLETE	MALE	1320	5	6	NICU	Y	-	-	-
53	1110016	11CM	-	-	Y	YES	NO	MALE	2450	9	9	ROOM	-	-	-	-
54	1109457	7CM	-	-	Y	YES	COMPLETE	MALE	2210	8	8	ROOM	-	Y	-	-
55	893558	1-2CM	-	-	Y	YES	COMPLETE	FEMALE	1420	6	6	NICU	Y	-	-	-
56	1015048	17CM	Y	-	-	YES	NO	FEMALE	2390	8	9	ROOM	-	-	-	-
57	1002004	NIL	Y	-	-	YES	INCOMPLETE	FEMALE	1280	5	6	NICU	Y	-	-	-
58	870943	12CM	Y	-	-	YES	COMPLETE	MALE	1320	5	6	NICU	Y	-	-	-
59	667246	9CM	-	-	Y	YES	NO	FEMALE	2370	8	9	ROOM	-	Y	-	-
60	667092	9CM	Y	-	-	YES	COMPLETE	MALE	620	3	4	NICU	Y	-	-	YES
61	610294	8CM	-	-	Y	YES	COMPLETE	MALE	2000	8	8	ROOM	-	-	-	-
62	600323	10CM	-	-	Y	YES	COMPLETE	MALE	1680	6	7	NICU	-	-	Y	-
63	600018	9CM	Y	-	-	YES	NO	FEMALE	2420	9	9	ROOM	-	-	-	-
64	597987	1CM	Y	-	-	YES	COMPLETE	FEMALE	1550	6	6	NICU	-	-	Y	-
65	596107	13CM	-	-	Y	YES	COMPLETE	MALE	1610	6	7	ROOM	-	-	-	-
66	583451	6CM	Y	-	-	YES	COMPLETE	MALE	1250	5	6	NICU	Y	-	-	-
67	565679	1-2CM	-	-	Y	YES	NO	MALE	2480	9	9	ROOM	-	Y	-	-
68	565214	10CM	Y	-	-	YES	COMPLETE	FEMALE	1410	5	6	NICU	Y	-	-	-
69	554919	NIL	-	-	Y	YES	COMPLETE	FEMALE	1440	5	6	NICU	Y	-	-	-
70	549980	11CM	Y	-	-	YES	COMPLETE	MALE	1150	5	5	NICU	Y	-	-	-
71	543091	7CM	-	-	Y	YES	NO	MALE	2450	9	9	ROOM	-	-	-	-
72	500289	18CM	-	-	Y	YES	NO	FEMALE	2550	9	9	ROOM	-	-	-	-
73	487999	10CM	Y	-	-	YES	INCOMPLETE	FEMALE	1650	6	6	NICU	-	-	Y	-
74	478941	2CM	Y	-	Y	YES	NO	MALE	2208	8	8	ROOM	-	Y	-	-
75	467610	8CM	Y	-	-	YES	COMPLETE	MALE	1480	6	6	NICU	Y	-	-	-
76	1034403	9CM	Y	-	-	YES	INCOMPLETE	MALE	2050	8	8	ROOM	-	-	-	-

Sr. No	MRD	NAME	AGE	DOA	BOOKED / UNBOOKED	EDUCATION	SOCIO-ECONOMIC STATUS	PRESENTING COMPLAINTS			OBSTETRICS HISTORY		MULTI	PRIMI
								H/O AMENORRHEA	2. h/o LEAKING SINCE(HOURS)	3. ASSOCIATED LABOR PAINS SINCE	LMP	EDD		
77	673309	P	34	31/08/2020	BOOKED	O	LOWER MIDDLE	36WEEKS6DAYS	1	NO	17/12/19	22/9/20	-	Y
78	662948	S	32	29/8/2020	UNBOOKED	O	UPPER LOWER	27WEEKS4DAYS	24	24HOURS	18/2/20	24/11/20	-	Y
79	6876	A	35	23/7/2020	BOOKED	PG	UPPER LOWER	36WEEKS	3	2HOURS	14/11/19	20/8/20	Y	-
80	600021	H	34	1/7/2020	BOOKED	O	LOWER MIDDLE	27WEEKS6DAYS	4	NO	19/12/19	24/9/20	-	Y
81	600650	A	29	1/7/2020	BOOKED	G	UPPER MIDDLE	30WEEKS5DAYS	16	6HOURS	29/11/19	4/9/2020	Y	-
82	598362	C	28	17/6/2020	BOOKED	G	LOWER MIDDLE	31WEEKS	18	48HOURS	13/11/19	19/8/20	-	Y
83	590142	A	27	11/6/2020	BOOKED	G	UPPER MIDDLE	31WEEKS6DAYS	48	33HOURS	1/11/2019	7/8/2020	-	Y
84	589705	T	33	4/6/2020	BOOKED	PG	UPPER CLASS	33WEEKS6DAYS	14	NO	11/10/2019	17/7/20	Y	-
85	582102	R	20	2/6/2020	BOOKED	O	LOWER MIDDLE	26WEEKS6DAYS	26	24HOURS	27/11/19	2/9/2020	-	Y
86	582000	M	31	20/5/2020	BOOKED	O	UPPER MIDDLE	35WEEKS	72	48HOURS	18/9/19	24/6/20	-	Y
87	574693	G	34	16/5/2020	BOOKED	O	LOWER MIDDLE	33WEEKS4DAYS	5	NO	24/9/19	30/6/20	Y	-
88	571028	E	26	12/5/2020	BOOKED	G	UPPER CLASS	32WEEKS	48	48HOURS	1/10/2019	7/7/2020	Y	-
89	569983	I	35	11/5/2020	BOOKED	G	LOWER MIDDLE	33WEEKS6DAYS	18	NO	17/9/19	23/6/20	Y	-
90	565605	D	31	10/5/2020	BOOKED	O	UPPER CLASS	36WEEKS	2	NO	1/9/2019	7/6/2020	Y	-
91	557841	S	36	1/5/2020	BOOKED	O	LOWER MIDDLE	30WEEKS4DAYS	10	NO	30/9/19	6/7/2020	-	Y
92	8710	S	33	22/4/2020	BOOKED	G	UPPER CLASS	36WEEKS5DAYS	72	36HOUR	9/8/2019	15/5/20	-	Y
93	512010	S	26	20/4/2020	BOOKED	O	UPPER LOWER	32WEEKS1DAY	72	40HOURS	8/9/2019	14/6/20	Y	-
94	501838	B	32	17/4/2020	BOOKED	O	LOWER MIDDLE	33WEEKS6DAYS	10	NO	24/8/19	30/5/20	-	Y
95	500912	Y	27	9/4/2020	BOOKED	G	UPPER MIDDLE	32WEEKS6DAYS	48	32HOURS	23/8/19	29/5/20	-	Y
96	500334	P	34	1/4/2020	BOOKED	PG	UPPER CLASS	36WEEKS6DAYS	24	NO	18/7/19	23/4/20	Y	-
97	433453	S	35	29/2/2020	BOOKED	O	LOWER	34WEEKS2DAYS	24	NO	4/7/2019	9/4/2020	-	Y
98	421989	A	28	28/2/2020	BOOKED	G	UPPER MIDDLE	31WEEKS3DAYS	28	28HOURS	23/7/19	28/4/20	-	Y
99	421183	R	36	27/2/2020	BOOKED	O	UPPER LOWER	30WEEKS5DAYS	24	NO	27/7/19	2/5/2020	-	Y
100	412684	V	33	25/2/2020	BOOKED	PG	UPPER CLASS	36WEEKS2DAYS	30	30HOURS	16/6/19	22/3/20	-	Y

Sr. No	MRD	H/O OF PPROM IN PREVIOUS PREGNANCIES	GENERAL PHYSICAL EXAMINATION				COLOUR	INVESTIGATION					
			BMI	B.P	PR	TEMPERATURE		Hb%	TLC	CRP	URINE-R	HIGH VAGINAL SWAB C/S	URINE C/S
77	673309	-	20.3	110/70	98	98.4	MECONIUM	12.9	7.9	13.2	BACTERIA+	NO GROWTH	KLEBSIELLA PNEUMONIA 10^5
78	662948	-	23.4	120/70	86	98	CLEAR	13.2	15.6	7	NAD	NO GROWTH	NO GROWTH
79	6876	YES	21.1	110/70	90	98.4	CLEAR	9.6	8.9	2.3	6-7PUS CELLS	NO GROWTH	NO GROWTH
80	600021	-	20.9	110/72	84	101.2	MECONIUM	9	24	36	BACTERIA+	E.COLI ++	E.COLI 10^5
81	600650	NO	25	120/78	78	98.2	CLEAR	12	7.6	1.6	NAD	NO GROWTH	NO GROWTH
82	598362	-	21.3	110/76	80	98.6	CLEAR	11.2	8.1	2.2	NAD	NO GROWTH	NO GROWTH
83	590142	-	18	120/70	92	98	BLOOD TINGED	10.7	12.9	4.7	19-20PUS CELLS	NO GROWTH	NO GROWTH
84	589705	YES	19.8	110/70	88	97.4	CLEAR	12.5	7.2	2.7	NAD	E.COLI ++	NO GROWTH
85	582102	-	20.3	100/70	72	98	CLEAR	11.1	9.9	3.8	NAD	NO GROWTH	NO GROWTH
86	582000	-	22.6	110/70	80	97.6	CLEAR	10	7.7	7.5	BACTERIA+	NO GROWTH	PSEUDOMONAS AERUGINOSA 10^5
87	574693	YES	23.3	100/68	100	98.4	CLEAR	12.3	13.1	4.9	NAD	CANDIDA+++	NO GROWTH
88	571028	NO	19.6	120/80	84	98	CLEAR	11.7	8.9	0.7	NAD	NO GROWTH	NO GROWTH
89	569983	YES	22.5	110/74	108	99.8	CLEAR	9.8	17.3	26	BACTERIA+	NO GROWTH	E.COLI 10^5
90	565605	NO	18.7	120/80	88	98	BLOOD TINGED	12.8	6.7	1.6	NAD	NO GROWTH	NO GROWTH
91	557841	-	26.1	110/80	92	97.6	CLEAR	12.6	15.9	13.8	BACTERIA+	NO GROWTH	E.COLI 10^5
92	8710	-	21.1	110/70	106	99.4	CLEAR	10.7	19.1	6.6	BACTERIA+	NO GROWTH	ENTEROCOCCUS FAECALIS 10^5
93	512010	NO	22	100/60	78	98	CLEAR	11.4	10.7	6	NAD	NO GROWTH	NO GROWTH
94	501838	-	24.4	110/70	80	98.6	CLEAR	10.9	12	3.7	NAD	NO GROWTH	NO GROWTH
95	500912	-	20.8	110/70	86	97.8	BLOOD TINGED	11.8	9.7	4.4	NAD	E.COLI ++	NO GROWTH
96	500334	YES	19.7	100/60	76	98.6	MECONIUM	12.4	5.8	1.5	2-3PUS CELLS	NO GROWTH	NO GROWTH
97	433453	-	23.4	120/70	98	97.4	CLEAR	11.2	12.3	0.9	YEAST+	CANDIDA++	NO GROWTH
98	421989	-	22.4	110/70	68	98.2	CLEAR	12.5	8.3	2.6	NAD	NO GROWTH	NO GROWTH
99	421183	-	21.8	100/70	80	98.4	MECONIUM	11.4	9.7	3.1	NAD	NO GROWTH	NO GROWTH
100	412684	-	20.1	120/78	92	97.6	CLEAR	10.1	15.6	12.3	BACTERIA+	NO GROWTH	E.COLI 10^5

Sr. No	MRD	USG (AFI)	ACTIVE MANAGEMENT			ANTIBIOTICS	STEROIDS	FETAL OUTCOME				SHIFTED TO NICU / ROOMING	ASSOCIATED RESPIRATORY MORBIDITY			FETAL MORTALITY
			MODE OF DELIVERY					SEX	BIRTH WEIGHT(GRAMS)	APGAR SCORE						
			NORMAL	INSTRUMENTAL	LSCS					1 Min	5 Min		RDS	TTNB	PNEUMONIA	
77	673309	13CM	-	-	Y	YES	NO	FEMALE	2620	8	9	ROOM	-	-	-	-
78	662948	10CM	-	-	Y	YES	COMPLETE	FEMALE	960	5	6	NICU	Y	-	-	-
79	6876	6CM	-	-	Y	YES	NO	MALE	2520	9	9	ROOM	-	-	-	-
80	600021	2CM	-	-	Y	YES	INCOMPLETE	MALE	990	6	6	NICU	Y	-	-	-
81	600650	10CM	Y	-	-	YES	COMPLETE	FEMALE	1350	6	7	NICU	Y	-	-	-
82	598362	13CM	Y	-	-	YES	COMPLETE	MALE	1400	5	6	NICU	Y	-	-	-
83	590142	9CM	-	-	Y	YES	COMPLETE	MALE	1520	6	6	ROOM	-	-	Y	-
84	589705	12CM	-	-	Y	YES	INCOMPLETE	FEMALE	2040	7	8	ROOM	-	-	-	-
85	582102	10CM	Y	-	-	YES	COMPLETE	MALE	880	5	6	NICU	Y	-	-	-
86	582000	18CM	-	-	Y	YES	COMPLETE	FEMALE	2380	8	9	ROOM	-	Y	-	-
87	574693	6CM	Y	-	-	YES	COMPLETE	MALE	1980	7	8	ROOM	-	-	-	-
88	571028	16CM	Y	-	-	YES	INCOMPLETE	MALE	1550	6	7	ROOM	-	-	Y	-
89	569983	3CM	Y	-	-	YES	COMPLETE	FEMALE	2040	8	8	ROO M	-	-	-	-
90	565605	15CM	-	-	Y	YES	NO	MALE	2620	9	9	ROOM	-	-	-	-
91	557841	5CM	Y	-	-	YES	COMPLETE	FEMALE	1250	5	6	NICU	Y	-	-	-
92	8710	8CM	-	-	-	YES	NO	FEMALE	2540	8	9	ROOM	-	-	-	-
93	512010	17CM	-	-	Y	YES	COMPLETE	FEMALE	1580	5	6	NICU	-	-	Y	-
94	501838	11CM	Y	-	-	YES	COMPLETE	MALE	2060	7	8	ROOM	-	-	-	-
95	500912	12CM	-	-	Y	YES	COMPLETE	FEMALE	1620	6	6	NICU	-	-	Y	-
96	500334	14CM	-	-	Y	YES	NO	MALE	2700	9	9	ROOM	-	-	-	-
97	433453	2CM	Y	-	-	YES	NO	MALE	2210	8	8	ROOM	-	-	-	-
98	421989	12CM	Y	-	-	YES	COMPLETE	FEMALE	1500	5	6	NICU	-	-	Y	-
99	421183	10CM	-	-	Y	YES	COMPLETE	MALE	1410	5	6	NICU	Y	-	-	-
100	412684	7CM	Y	-	-	YES	NO	FEMALE	2690	9	9	ROOM	-	-	-	-

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The Principal,
DMC, Ludhiana

Subject: Approval of Plan of Thesis.

This is to inform you that the Faculty of Medical Sciences in its meeting held on 22.01.2021 has approved the plan of thesis of **Dr. Riddhima Gupta,** a student of your college for **MD Obstetrics & Gynaecology** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
To study the relationship of amniotic fluid volume with neonatal respiratory morbidity

Supervisor
Dr. Pooja Tandon
Assoc. Prof.,
Dept. of Obst. & Gynae,
DMCH, Ludhiana.

Co-Supervisor
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Dr. Kamaldeep Arora
Assoc. Prof.,
Dept. of Paediatrics,

The student has to submit the three copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

S P Singh

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. ***If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.***
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

TO STUDY THE RELATIONSHIP OF AMNIOTIC FLUID VOLUME WITH NEONATAL RESPIRATORY MORBIDITY

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN

PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MD

In

OBSTETRICS & GYNAECOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

NOVEMBER, 2019

DR. RIDDHIMA GUPTA

DEPARTMENT OF OBSTETRICS & GYNAECOLOGY
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	TO STUDY THE RELATIONSHIP OF AMNIOTIC FLUID VOLUME WITH NEONATAL RESPIRATORY MORBIDITY
For the degree of	MD (OBSTETRICS & GYNAECOLOGY)
Name of the candidate	Dr. Riddhima Gupta
Supervisor	Dr. Pooja Tandon, Associate Professor, Department of Obstetrics & Gynaecology, DMCH, Ludhiana
Co-supervisor(s)	Dr. Ashima Taneja, Professor & Head, Department of Obstetrics & Gynaecology, DMCH, Ludhiana Dr. Kamaldeep Arora, Associate Professor, Department Of Pediatrics, DMCH
Institution	Dayanand Medical College and Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: PPROM is defined as preterm premature rupture of membranes prior to 37 weeks completed gestation. It occurs in 2 % to 4 % of all singleton and 7 % to 20 % of twin pregnancies.

Aim of Study: To study the association of Amniotic fluid volume at presentation in patients with preterm premature rupture of membranes, with neonatal respiratory morbidity.

Materials & Methods: A Hospital based, time bound PROSPECTIVE clinical study will be conducted in the department of Obstetrics and Gynaecology at Dayanand Medical College and Hospital (Ludhiana) for a time period of 1.5 years, from Jan 1st 2020 to June 30th 2021.

Clinical Significance: PPROM remains a challenge for both obstetricians and neonatologists. Despite improvements in neonatal outcome, mortality and morbidity remain high, and there are few long-term data for infants born in this context. Further research is needed in order to understand the pathophysiological mechanisms causing neonatal pulmonary morbidity.

Keywords: PPROM-preterm premature rupture of membranes, Oligohydramnios, Neonatal respiratory morbidity

(Signature)	(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"TO STUDY THE RELATIONSHIP OF AMNIOTIC FLUID VOLUME WITH NEONATAL RESPIRATORY MORBIDITY"** exist in the Department of Gynaecology, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate herself under our supervision and guidance.

Supervisor

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Place : _____

Date : _____

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Riddhima Gupta
Department	Obstetrics & Gynaecology
Topic of Thesis	TO STUDY THE RELATIONSHIP OF AMNIOTIC FLUID VOLUME WITH NEONATAL RESPIRATORY MORBIDITY
Likely date of appearing in PG examination	June 2022
Date of enrollment	16th May, 2019
Name of Head of Department	Dr. Ashima Taneja
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2) Co-Supervisor(s)	Dr. Ashima Taneja Dr. Kamaldeep Arora
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Jaspal Singh (Professor & Head, Surgery)	Mr. A K Jindal (Member, Ethics Committee)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Assistant Dean Academics)	Dr. Deepinder K Chhina (Professor & Head, Microbiology)
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Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / ~~No~~

Dr. Sandeep Puri
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Research Committee

Chairperson
Research Committee

Approved: Yes / ~~No~~

Prof. Arvind Malhotra
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Ethics Committee

Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	DR RIDDHIMA GUPTA 27 CIRCULAR ROAD NEAR MEDICAL ENCLAVE AMRITSAR PUNJAB
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL ,LUDHIANA
3	COURSE OF STUDY AND SUBJECT	MD OBSTETRICS AND GYNAECOLOGY
4	DATE OF ADMISSION OF COURSE	16 May 2019
5	TITLE OF THE TOPIC	TO STUDY THE RELATIONSHIP OF AMNIOTIC FLUID VOLUME WITH NEONATAL RESPIRATORY MORBIDITY

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

The etiology of PPROM is multifactorial, although intrauterine infection is the most common risk factor, especially at an earlier gestational age.[1] Moreover, women with PPROM and preterm labor often have highly positive cultures; infection preceding PPROM is often subclinical and thought to ascend from the lower genital tract.[2-4] This ascending bacterial invasion may lead to intrauterine infection in up to 60% of cases with PPROM.[5]

One of the major functions of fetal membranes is to protect the fetus during its growth and development. Moreover, they provide mechanical and immune protection and act as a barrier for microbial access.[6,7] Compromise in the immune and mechanical properties of the fetal membranes allows for microbial invasion from the genital tract, activation of host inflammatory response leading to collagenolysis mediated mechanical disruption, and membrane weakening leading to PPROM.[8,9]

PPROM is an important cause of perinatal morbidity and mortality, which is mainly due to prematurity as well as risks associated with expectant management of PPROM including perinatal infection, placental abruption, umbilical cord prolapse, and umbilical cord compression in the presence of oligohydramnios.[10,11]

It has been postulated that fetal lung development is dependent on adequate amount of amniotic fluid, and that fetal exposure to severe oligohydramnios during critical periods of gestation may result in pulmonary hypoplasia. Although pulmonary hypoplasia has been mainly associated with severe oligohydramnios in early gestation.[12,13]

Fetal morbidity after PPROM results from maternal intrauterine infection, umbilical cord compression, placental abruption, and prolonged fetal compression caused by oligohydramnios. Each of these places the fetus at increased risk for fetal death (generally approximately 1% with conservative management after the limit of potential neonatal viability) and perinatal asphyxia. The pregnancy

complicated by PPROM before the limit of fetal viability (currently b23 weeks) is at increased risk for fetal demise (15%); however, a portion of this increase is attributable to nonintervention for fetal benefit when delivery occurs before there is any hope of postnatal survival. When membrane rupture occurs well before the limit of fetal viability (particularly when there is persistent oligohydramnios), there is a significant risk of lethal fetal pulmonary hypoplasia caused by arrested alveolar development. This becomes evident with failure of lung growth despite prolonged latency. Prolonged compression can lead to fetal restriction deformities, similar to those seen in Potter's syndrome. There is accumulating evidence that in utero exposure to infection increases the risk of long-term neurologic sequelae [14], although there are not current data to demonstrate that delivery before the onset of clinical symptoms of infection prevents these adverse outcomes. The primary determinant of infant morbidity and mortality is gestational age at delivery. In general, infant morbidity can be anticipated to be similar to that of other infants born at the same gestational age (absent pulmonary hypoplasia). Umbilical cord compression before and during labor and placental abruption, however, theoretically increase this risk of hypoxic insult. Additionally, the risk of neonatal infection is approximately twofold higher at any gestational age when delivery occurs after preterm PROM than for other causes. Group B streptococcus is a significant cause of early onset neonatal sepsis and is more likely to preterm premature rupture of the membranes 413 occur in the setting of premature birth, prolonged membrane rupture, or amnionitis, each of which is seen commonly with preterm PROM. Lethal pulmonary hypoplasia is rare with PROM, occurring after 24 to 26 weeks' gestation, presumably because alveolar development by this time is adequate to support postnatal life. With PPROM remote from term, however, there is the potential for nonlethal pulmonary hypoplasia, manifesting through postnatal pulmonary complications including pneumothorax, pneumomediastinum, and the need for high ventilatory pressures to compensate for poor pulmonary compliance. In general survival is likely and long-term sequelae uncommon with

delivery at or after 32 weeks' gestation (unless PPROM occurs before 24–26 weeks' gestation). This is not a distinct cutoff, but rather a continuum with some infants doing well despite earlier delivery and a small number of infants having poor outcomes with delivery near term. Thus, it is important to find out the relation between oligohydramnios due to PPROM and its effect on neonatal respiratory morbidity.

6.2 REVIEW OF LITERATURE

Amniotic membranes usually rupture at the time of labor but sometimes the membranes may rupture before the onset of the labor and this is called as Premature rupture of membranes (PROM). According to William's, PROM is defined as rupture of membranes before the onset of labor. When it occurs before 37 completed weeks it is called as " preterm premature rupture of membranes (pPROM)" and when it occurs after 37 completed weeks it is " term PROM".[15]

The fetal membranes insulate the fetus and the amniotic fluid against microbial infections. Together with the amniotic fluid the membranes protect the fetus against blunt trauma and the umbilical cord against compression. In addition the membranes have the role in the enzymatic degradation of the uterotonic substances produced in the chorionic decidual interphase. With its multiple functions it is not surprising that the PPROM can cause potentially severe adverse effects in pregnancies.[16]

Harger et.al. and Asrat showed a strong association between previous h/o PPROM and recurrence of PPROM.[17]

Asrat et.al reported a 32% recurrence risk of PPROM in patients with a previous history of PPROM.[17]

Nelson et.al. reported that the leakage of fluid in PPROM could lead to a disproportion between the placental and uterine surface areas, thus favoring placental separation. It may be secondary to chronic uterine decompression. Vintziloes et.al. demonstrated that placental abruption was common in patients with PPROM and oligohydramnios.[17]

The patients with PPROM have a high rate of caesarean section rate than uncomplicated pregnancies (8.8% v/2.1%).[17]

Histological chorioamnionitis complicates almost half of all PPROM cases that occur prior to 34 weeks' gestation.[18] Yu et al. published a report of pregnancies with PPROM at <34 weeks and noted a rate of chorioamnionitis to be around 17.8%.[19] The latency period exceeded 7 days in only 24.3% of cases.

Paolo Venturini conducted a study "Induction of labor in women with oligohydramnios" to evaluate the outcome of labor induction in women with
oligohydramnios at term. This was a prospective case-control study which included 120 consecutive patients with oligohydramnios undergoing labor induction. One hundred and sixteen patients with normal amniotic fluid matched for gestational age and Bishop-score served as controls. They concluded that Oligohydranmios at term did not influence the outcome of induction of labour in nulliparous women with unfavorable cervix.[20]

Vidya A. Thobbi and Sheema Sabahath conducted a study upon 200 cases of isolated oligohydramnios and concluded that in primis with oligo were 48%.

NST was reactive in 62%.USG profile being normal in 80% and 20% abnormal. Out of these, 25% had vaginal delivery and 75% CS. Fetal distress (62%) being the major indication for CS.58% of neonates had a mean birth wt of >2.5 kg, 38% had NICU admission. Perinatal mortality observed in 12%. They concluded that oligohydramnios is associated with LBW, low APGAR score and higher NICU admission. Pregnancy with isolated oligohydramnios is not associated with impaired fetal growth or an increased risk of adverse perinatal outcome and requires no aggressive treatment.[21]

It is well established that fetal lung development is dependent on adequate amount of amniotic fluid, and that fetal exposure to severe oligohydramnios during critical periods of gestation may result in pulmonary hypoplasia. Although pulmonary hypoplasia has been mainly associated with severe oligohydramnios in early gestation, there is insufficient data as to whether this association extends to severe oligohydramnios during late-second or early third trimester, as well as to the degree of oligohydramnios that carries respiratory consequences.[22]

Combs C. Andrews MD, et.al. (2004) concluded in their studies that aggressive tocolysis caused significant maternal complications after

PPROM and did not prolong pregnancy compared with no tocolysis at all.[23]

Ananth, Cande, V. et.al. in Meta analysis indicate that the antibiotics therapy in patients with PPROM improves the outcome for both mother and baby. Mothers have fewer episodes of infection.Infants have a prolonged latency phase and less likely to suffer a perinatal death. The risk of neonatal sepsis is not increased after antibiotic exposure, this silencing the fear that the treatment would select out resistant organisms.[24,25]

Clinical presentation of neonatal respiratory distress involves tachypnea (rate>60 breaths per minute), cyanosis, expiratory grunting with chest retractions, and nasal flaring. The underlying disease may be due to pulmonary, cardiac, infectious, metabolic, or other systemic disorders. Peripheral cyanosis or acrocyanosis is often observed in normal newborn infants or in ill infants with poor cardiac output. Central cyanosis is assessed by examining the oral mucosa and suggests inadequate gas exchange signifying more than 3 to 5 g/dL of desaturated hemoglobin. Clinical determination of central cyanosis may be unreliable(ie, not observed) in severely anemic patients despite low PaO2 values; in contrast, polycythemic infants may appear cyanotic despite normal values of PaO2. Hence, oxygen saturation measured by pulse oximetry (arterial oxygen saturation [Sao2]) is recommended by the American Academy of Pediatrics to screen infants for hypoxemia, and Sao2 values less than 90% after 15 minutes of age are considered abnormal.(26)Decrease in O2 saturation, apnea, or both may be present in infants with respiratory distress.[26-27] Irregular (seesaw) or slow respiratory rates of less than 30 breaths per minute if associated with gasping may be an ominous sign. Chest retractions occur because the neonatal chest wall is compliant making it susceptible to alterations in lung function resulting in substernal, subcostal, or intercostal retractions. The retractions result from negative intrapleural pressure generated by the contraction of diaphragm and accessory chest wall muscles along with impaired mechanical properties of the lungs and chest wall. Retractions are

observed in lung parenchymal diseases such as respiratory distress syndrome (RDS), pneumonia, airway disorders, pneumothorax, atelectasis, or bronchopulmonary dysplasia (BPD). Nasal flaring is caused by contraction of alae nasi muscles decreasing the resistance in the nares with resultant reduced work of breathing. Neonates primarily breathe through the nose, hence nasal resistance contributes significantly to total lung resistance, which occurs in choanal atresia or obstruction due to secretions. During resuscitation, suction of mouth is followed by suctioning the nose to prevent aspiration of secretions, blood, or meconium. Occasionally, nasal flaring is observed during feeding or active sleep in normal infants. Grunting is a compensatory effort made during expiration by closure of the glottis, increasing the airway pressure and lung volume with resultant increased ventilation perfusion (V/Q) ratio. Unlike normal breathing, wherein the vocal cords abduct to enhance inspiratory flow, expiration through partially closed vocal cords in some respiratory disorders produces a grunting sound. Depending on the severity of lung disease, grunting may be either intermittent or continuous. Grunting can maintain FRC and values of PaO2 equivalent to those during the application of 2 or 3 cm H2O of continuous distending pressure. Accessory respiratory muscles also assist in optimizing upper airway functions. The genioglossus muscle protrudes the tongue and maintains pharyngeal patency, whereas the laryngeal muscles move the vocal cords regulating airflow during expiration.

Nicolini et al[28] have hypothesized that lung hypoplasia is due to a low amniotic fluid pressure. The normal intra-amniotic pressure ranges between 1-14 mm Hg. However,with oligohydramnios and intact membranes the intra-amniotic pressure </= 1 mmHg. There is an increase in intra-alveolar — amniotic fluid gradient resulting in a greater outflow of lung fluid. When there is a sufficient loss of intra-alveolar fluid, lung growth is impaired.

6.3 AIMS AND OBJECTIVES OF THE STUDY

- To determine the relationship between amniotic fluid volume in patients with preterm premature rupture of membranes and its effect on neonatal respiratory morbidity.

7. MATERIALS AND METHODS

A Hospital based, time bound PROSPECTIVE clinical study will be conducted in the department of Obstetrics and Gynaecology at Dayanand Medical College and Hospital (Ludhiana) for a time period of 1.5 years, from Jan 1st 2020 to June 30th 2021.

7.1 Source of Data

Patients admitted with preterm premature rupture of membranes between 23-37 weeks of gestation at Dayanand Medical College and Hospital, Ludhiana.

7.2 METHOD OF COLLECTION OF DATA

The study will consist of prospective study of singleton pregnancies with PPROM. All mothers and babies will be observed from the time of admission to the time of discharge.

INCLUSION CRITERIA:

All pregnant women with a singleton pregnancy between 23-37 weeks of gestational age with preterm premature rupture of membranes.

EXCLUSION CRITERIA:

- Multiple pregnancies
- Intrauterine growth restriction
- Uterine anamolies
- Fetal anamolies
- Myoma uteri
- Hypertensive disorders and pregnancy induced hypertension
- Gestational diabetes mellitus
- Antepartum haemorrhage
- Chronic renal failure
- Class II to IV cardiac diseases

METHODOLOGY:

Data to be collected noting the following:-

A written and informed consent will be taken from all the subjects entering into the study. A detailed history will be taken and thorough clinical examination will be carried out. Obstetric examination will be

done that will include per abdomen examination for labor assessment, pelvic examination for bishop scoring and pelvic assessment. NST recording for 20 mins on admission. An ultrasound examination for fetal assessment, amount of liquor (AFI) within 7 days of delivery.

Detailed history regarding the age, parity, socio economic status and previous obstetric history will be noted. The exact date of last menstrual period will be noted. The cases will be labeled as having PPROM depending on history and examination. Detailed antenatal history will be taken with particular attention to previous obstetric history.

A full clinical examination would be carried out particularly regarding pulse, BP, abdominal palpation to confirm presentation, uterine contraction and fetal heart rate.

Per speculum examination to see the presence of pool of liquor in the posterior fornix or coming from the cervix to confirm the diagnosis. Per vaginal examination will be be done if the patient is in labor with aseptic precautions to confirm the presentation, cervical dialatation and effacement.

Investigations like Hb %, TC, DC, CRP, urine routine, urine culture and sensitivity and cervical swab for C/S will be sent. The latent period, duration of stages of labor, induction to delivery interval and PPROM to delivery interval will be noted. Maternal pulse, BP and fetal heart rate will be checked frequently.

Soon after delivery Apgar score at 1 and 5 minutes, six, birth weight, congenital anomalies, immediate complications like birth injuries, signs of asphyxia, meconium aspiration, sepsis and other associated complications will be recorded. In the presence of complications, babies admitted to NICU in the postnatal period will be followed up. Neonatal morbidity and mortality will be noted.

OUTCOME:

Association between Oligohydramnios due to ,PPROM and neonatal respiratory morbidity.

STATISTICAL ANALYSIS:

Data will be described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

1. Blood group and Rh typing
2. Hemoglobin%
3. Coagulation profile
4. Urine routine examination
5. Urine culture & sensitivity
6. High vaginal swab
7. CRP(quantitative)
8. Ultrasound for fetal well being and AFI

All the above mentioned investigations are routinely performed as per the hospital guidelines. Hence will not pose any financial burden on the patient.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith

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PROFORMA

Sr. No

NAME: I.P.NO:

AGE: UNIT:

ADDRESS: DOA:

DOD:

OCCUPATION: BOOKED / UNBOOKED:

RELIGION: EDUCATION : H -

SOCIO-ECONOMIC STATUS: W -

PRESENTING COMPLAINTS:

1. h/o AMENORRHEA:
2. h/o DRAINING SINCE: Hrs
3. ASSOCIATED LABOR PAINS SINCE: Hrs

OBSTETRICS HISTORY

MARRIED LIFE- CONSANGUINITY -

G---------- P ---------- L ---------- A----------

LMP

EDD

PREVIOUS PREGNANCIES

No. of preg.	Delivery Outcome	Puerperium
1		
2		
3		
4		

PRESENT PREGNANCY

I TRIMESTER

II TRIMESTER

III TRIMESTER

H/O OF PPROM IN PREVIOUS PREGNANCIES --PRESENT/ABSENT

L C B

MENSTRUAL HISTORY

MENARCHY: CYCLES:

LMP:

EDD:

PAST HISTORY:

FAMILY HISTORY:

PERSONAL HISTORY:

GENERAL PHYSICAL EXAMINATION.

BUILT: PALLOR:

NOURISHMENT: P. EDEMA:

HEIGHT: HYDRATION:

WEIGHT:

PULSE: THYROID:

B.P: BREAST:

TEMPERATURE: SPINE:

R.R:

SYSTEMIC EXAMINATION

R.S.:

C.V.S:

P.A INSPECTION:

PALPATION:

HT. OF UTERUS: FUNDAL GRIP: UMBLICAL GRIP: 1ST PELVIC GRIP:

2nd PELVIC GRIP:

FETAL HEART SOUND

UTERUS RELAXED / IRRITABLE / ACTING : SFH :

LIE : PRESENTATION :

POSITION:

ATTITUDE :

LIQUOR :

UTERINE TENDERNESS :

LIQUOR COLOUR ODOUR

PELVIC EXAMINATION :

BISHOP SCORES : P/V

INVESTIGATION : Hb% :

TC :

DC :

CRP :

BLOOD GROUP /RH TYPING : URINE-R :

HIGH VAGINAL SWAB C/S :

URINE C/S :

USG :

MODE OF TREATMENT :

CONSERVATIVE : MATERNAL & FETAL MONITORING :

Time	Pulse	BP	Temp	PA	FHS	PV	Treatment

ACTIVE MANAGEMENT: SPONTANEOUS DELIVERY :

INDUCTION : OXYTONIC / PG :

ANTIBIOTICS : STEROIDS :

LABOUR STARTED AT : DURATION OF 1ST STAGE : DURATION OF 2ND STAGE :

MODE OF DELIVERY : NORMAL / INSTRUMENTAL / LSCS :

LSCS : INDICATIONS :

PPROM TO DELIVERY INTERVAL : INDUCTION TO DELIVERY INTERVAL :

DURATION OF 3RD STAGE :

PLACENTA : HEALTHY / UNHEALTHY WEIGHT :

CALCIFICATION : INFECTION :

DRUGS USED IN 3RD STAGE : OXYTOCIN

METHERGIN

COMPLICATIONS OF 3RD STAGE : PPH :

RETAINED PLACENTA : OTHERS :

FETAL OUTCOME :

DATE TIME :

SEX : BIRTH WEIGHT :

APGAR SCORE : 1 Min.

5 Min.

RESUSCITATION : SUCTION / STIMULATION / OXYGEN BY MASK / FREE FLOW OXYGEN / IPPR/ TRACHEAL INTUBATION + SUCTION.

DRUGS : ANOMALIES

SHIFTED TO NICU / ROOMING

BALLARDS SCORE :

PUERPERAL PERIOD :

Day DATE TEMP PULSE BP BREAST RS/CVS PA LOCHIA/EPI CMT Treatment

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____
7. _____

BABY – INDICATION FOR SHIFTING TO NICU

DaY PROB CVS RS PA CNS MGN IVF TUBE FEED DRUGS

_____ 1.

_____ 2.

_____ 3.

_____ 4.

_____ 5.

_____ 6.

_____ 7.

_____

CONDITION OF MOTHER & BABY AT THE TIME OF DISCHARGE:

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....
.....am willing to participate in the study **"TO STUDY THE RELATIONSHIP OF AMNIOTIC FLUID VOLUME WITH NEONATAL RESPIRATORY MORBIDITY"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

.....
Signature of the Witness

.....
Signature of the patient

Name:

Signature of Candidate

Signature of Supervisor

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"AMNIOTIC FLUID VOLUME AT PRESENTATION WITH PRETERM PREMATURE RUPTURE OF MEMBRANES AND THE ASSOCIATION WITH NEONATAL RESPIRATORY MORBIDITY"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर